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4. CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter briefly summarizes the PM risk analyses conducted for two urban study areas (Philadelphia and Los Angeles counties) during the previous review of the PM NAAQS and describes the proposed scope of EPA's updated risk analyses to be conducted for the current review of the standards. The updated risk analyses will focus on the risks of mortality, morbidity, and symptoms associated with recent ambient air quality levels and just attaining the current suite of $PM_{2.5}$ NAAQS and any other alternative $PM_{2.5}$ standards that may be identified as appropriate for consideration during the course of the current review of the PM NAAQS. EPA also is considering the appropriateness of conducting risk analyses for respiratory-related hospital admissions and respiratory symptoms associated with coarse-fraction PM (i.e., $PM_{10-2.5}$) for recent air quality levels and upon just meeting potential $PM_{10-2.5}$ standards. Results from the updated risk analyses will be presented in the next draft of this Staff Paper. As discussed in Chapters 2, the fact that the sources and composition of $PM_{2.5}$ and $PM_{10-2.5}$ are largely distinct, along with the new health effects evidence discussed in Chapter 3, supports the recommendation from the previous Staff Paper that fine-and coarse-fraction particles be considered as separate pollutants. At that time, a number of health studies indicated differences in health effects between fine-and coarse-fraction particles, and suggested that serious health effects, such as premature mortality, were more closely associated with fine-fraction particles. The new studies, summarized in the draft CD (CD, Chapter 6), continue to show associations between serious health effects, including premature mortality, and fine-fraction PM, but they also offer new evidence indicating possible associations between coarse-fraction PM and health effects. For coarse-fraction particles the strongest evidence is found relating $PM_{10-2.5}$ ambient concentrations and increased respiratory hospital admissions and respiratory symptoms.

4.1.1 Goals for Updated PM Risk Analyses

The goals of the updated PM risk analyses are: (1) to provide a rough sense of the potential magnitude of PM-associated mortality and morbidity associated with current $PM_{2.5}$

levels and with attaining the current suite of $PM_{2.5}$ NAAQS (as well as any potential alternative $PM_{2.5}$ standards identified as part of this review); (2) to provide a rough sense of the potential magnitude of PM-associated morbidity associated with current $PM_{10-2.5}$ levels and with attaining possible alternative $PM_{10-2.5}$ NAAQS (if the decision is made that there is sufficient evidence to warrant conducting a risk analysis for coarse-fraction PM); (3) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates; and (4) to gain qualitative insights into the nature of the risks associated with exposure to PM. The staff recognizes that due to the many sources of uncertainty inherent in conducting PM risk analyses, any PM risk estimates presented in the next draft Staff Paper should not be interpreted as demonstrated health impacts or precise measures of risk. Further, the staff recognizes the limited role of the risk analyses in this standards review and do not plan to use the risk estimates as a principal basis for recommending selection among alternative standard levels.

4.1.2 Summary of Risk Analyses Conducted During Prior PM NAAQS Review

For the prior review, EPA conducted a number of risk analyses that estimated population risk for two defined urban study areas (i.e., Philadelphia and Los Angeles counties). The PM health risk model combined information about daily PM air quality for these two study areas with estimated concentration-response functions derived from epidemiological studies and baseline health incidence data for specific health endpoints to derive estimates of the annual incidence of specific health effects occurring under "as is" air quality. Since site-specific relative risks were not available for all endpoints in both locations (and in the absence of more information concerning which individual studies might best characterize the health risk in a given location), a form of meta analysis (referred to as a "pooled analysis") was conducted which combined the results of the studies that met specified criteria. The analyses also examined the reduction in estimated incidence that would result upon just attaining the existing PM_{10} standards and several sets of alternative $PM_{2.5}$ standards. The methodological approach followed in conducting the prior risk analyses is described in Section 6 of the 1996 Staff Paper (EPA, 1996b) and in several technical reports (Abt Associates, 1996; Abt Associates, 1997a,b) and articles (Post et al., 2000; Deck et al., 2001).

Summarized below are the key observations resulting from the prior risk analyses which were most pertinent to the decision on the PM NAAQS, as well as several important caveats and limitations associated with these analyses:

- EPA placed greater weight on the overall conclusions derived from the health effect studies – that PM air pollution is likely causing or contributing to significant adverse effects at levels below those permitted by the existing PM_{10} standards – than on the specific concentration-response functions and quantitative risk estimates derived from them. The quantitative risk estimates included significant uncertainty and, therefore, were not viewed as demonstrated health impacts. Nevertheless, EPA did state that it believed the analyses presented reasonable estimates as to the possible extent of risk for these effects given the available information (62 FR 38656).

- Consideration of key uncertainties and alternative assumptions resulted in fairly wide ranges in estimates of the incidence of PM-related mortality and morbidity effects and risk reductions associated with attainment of alternative standards in both locations in the risk analyses. Significantly, the combined results for these two cities alone found that the risk remaining after attaining the current PM_{10} standards was on the order of hundreds of premature deaths each year, hundreds to thousands of respiratory-related hospital admissions, and tens of thousands of additional respiratory-related symptoms in children (62 FR 38656).

- Based on the results from the sensitivity analyses of key uncertainties and the integrated uncertainty analyses, the single most important factor influencing the uncertainty associated with the risk estimates was whether or not a threshold concentration exists below which PM-associated health risks are not likely to occur (62 FR 38656).

- Over the course of a year, the few peak 24-hour $PM_{2.5}$ concentrations appeared to contribute a relatively small amount to the total health risk posed by the entire air quality distribution as compared to the aggregated risks associated with the low to mid-range $PM_{2.5}$ concentrations (62 FR 38656).

- There was greater uncertainty about both the existence and the magnitude of estimated excess mortality and other effects associated with $PM_{2.5}$ exposures as one considered lower concentrations that approach background levels (62 FR 38656).

- Based on the results from the sensitivity analyses of key uncertainties and/or the integrated uncertainty analyses, the following uncertainties had a much more modest impact on the risk estimates: inclusion of individual copollutant species when estimating PM effect sizes; the choice of approach to adjusting the slope in analyzing alternative cutpoints; the value chosen to represent average annual background PM concentrations; and the choice of rollback adjustment approaches for simulating attainment of alternative $PM_{2.5}$ standards (EPA, 1996b).

4.2 GENERAL SCOPE OF PLANNED PM RISK ANALYSES

As discussed in Chapter 3 above, the draft CD (CD, p. 9-40) finds that "[t]he newer experimental evidence, therefore, adds considerable support for interpreting the epidemiologic findings . . . as being indicative of causal relationships between exposures to ambient PM and consequent associated increased morbidity and mortality risks." The risk analyses planned for this NAAQS review are premised on the assumption that $PM_{2.5}$ is causally related to the mortality, morbidity, and symptomatic effects (alone and/or in combination with other pollutants) observed in the epidemiological studies. Since the last review, additional studies have been published which strengthen the basis for concern about mortality and morbidity health endpoints being related to ambient $PM_{2.5}$ exposures. Therefore, EPA plans to conduct risk analyses for $PM_{2.5}$ and several health endpoints, including mortality, hospital admissions, and respiratory symptoms. In addition, there is a growing, but limited data base reporting health effects associated with coarse-fraction PM and which uses $PM_{10-2.5}$ as the air quality indicator. The strongest evidence indicating potential health effects associated with coarse-fraction PM is for respiratory-related hospital admissions and respiratory symptoms. Currently, EPA is considering whether to conduct risk analyses for $PM_{10-2.5}$ for these two categories of effects.

The staff welcomes CASAC and public input on (1) the relevant health studies to include in the $PM_{2.5}$ risk analysis, (2) whether or not to conduct a limited coarse-fraction risk analysis, and (3) if a coarse-fraction risk analysis is conducted, which health endpoints and studies should be considered. The discussion below includes information on studies and concentration-response functions for both $PM_{2.5}$ and $PM_{10-2.5}$ to help inform a decision on whether to proceed with a limited coarse-fraction risk analysis focused on respiratory-related hospital admissions and respiratory symptoms. Similarly, air quality information on $PM_{10-2.5}$ for possible urban counties that could be selected for such analyses also are included in this chapter.

The planned $PM_{2.5}$ risk analyses will focus on selected health endpoints such as increased daily mortality, increased hospital admissions for respiratory and cardiopulmonary causes, and increased respiratory symptoms for children. A consequence of limiting the analyses to selected health endpoints is that the risk estimates may understate the type and extent of potential health

impacts of PM exposures. Although the risk analyses will not address all health effects for which there is some evidence of association with exposure to PM, all such effects are identified and considered in Chapter 3.

The risk assessment to be conducted as part of this review, like the prior risk assessment done as part of the last review (EPA, 1996b), will use concentration-response functions from epidemiological studies based on ambient PM concentrations measured at fixed-site, population-oriented, ambient monitors. As discussed earlier in Chapter 2 (Section 2.8), measurements of daily variations of ambient $PM_{2.5}$ concentrations, as used in the time-series studies that provide the concentration-response relationships for these analyses, have a plausible linkage to the daily variations of exposure from ambient sources for the populations represented by ambient monitoring stations. The draft CD concludes that this linkage is better for indicators of fine particles (e.g., $PM_{2.5}$) and PM_{10} but that this may not be the case for $PM_{10-2.5}$, for specific chemical components, for source contributions, or for sites located near sources (CD, p. 9-24). A more detailed discussion of the possible impact of exposure misclassification on the estimated concentration-response relationships derived from the community epidemiological studies is presented above in Chapter 3 (see Section 3.5.3.3).

While quantitative estimates of personal or population exposure do not enter into derivations of the risk estimates, an understanding of the nature of the relationships between ambient PM and its various components and human exposure underlies the conceptual basis for the risk assessment. Unlike recent reviews for ozone and carbon monoxide, where exposure analyses played an important role, a quantitative exposure analysis will not be conducted as part of this review since the currently available epidemiology health effects evidence relates ambient PM concentrations, not exposures, to health effects. As discussed in Chapter 4 of the draft CD, EPA and the exposure analysis community are working to improve exposure models designed specifically to address PM. Both EPA and the broader scientific community also are in the process of collecting new information in PM exposure measurement field studies that will improve the scientific basis for exposure analyses that may be considered in future reviews.

While the NAAQS are intended to provide protection from exposure to ambient PM, EPA recognizes that exposures to PM from other sources (i.e., non-ambient PM) also have the

potential to affect health. The EPA's Office of Radiation and Indoor Air and other Federal Agencies, such as the Consumer Product Safety Commission (CPSC) and the Occupational Safety and Health Administration (OSHA), address potential health effects related to indoor, occupational, environmental tobacco smoke, and other non-ambient sources of PM exposure. Like the prior risk analysis, contributions to health risk from non-ambient sources are beyond the scope of the proposed risk analyses for the NAAQS review.

This proposed PM health risk analysis is similar in many respects to the prior risk analysis conducted for the last PM NAAQS review. Both the prior and the current proposed PM risk analyses:

- estimate risks for the urban centers of example cities, rather than attempt a nationwide analysis.

- analyze risks under a recent 12-month period of air quality (labeled "as is") and under a situation where air quality just attains the current set of standards. (The risk analyses also will include any potential alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards that are identified as part of this review).

- estimate risks only for concentrations exceeding estimated background levels.

- present qualitative and quantitative considerations of uncertainty, including sensitivity analyses of key individual uncertainties and integrated sensitivity analyses combining key parameters.

Both the prior and the current planned PM risk analyses focus on health endpoints for which concentration-response functions have been estimated in epidemiological studies. Since these studies estimate concentration-response functions using air quality from fixed-site, population-oriented monitors, the appropriate application of these functions in a PM risk analysis similarly requires the use of air quality data from fixed-site, population-oriented, ambient monitors. This is identical to the approach taken in the last PM NAAQS review.

The scope of the planned $PM_{2.5}$ risk analyses is to develop risk estimates for at least two selected urban areas: Philadelphia County, and a portion (roughly the southeastern third) of Los Angeles County (hereafter referred to as "Los Angeles County"). The staff is soliciting comment on whether it should also include Salt Lake County in the $PM_{2.5}$ risk analyses, if it proceeds to conduct a coarse fraction PM analysis for this county. The scope of the potential $PM_{10-2.5}$ risk

analyses is to develop risk estimates for Los Angeles County and Salt Lake County. These areas have been chosen based on availability of $PM_{2.5}$ and $PM_{10-2.5}$ air quality data. There also is a desire to include areas from the eastern and western parts of the United States to reflect regional differences in the composition of $PM_{2..5}$. Because elevated $PM_{10-2.5}$ levels are primarily a problem in the western parts of the United States and because of the lack of eastern sites with adequate $PM_{10-2.5}$ data, EPA is considering conducting the potential coarse-fraction risk analyses only in the two western areas (i.e., Salt Lake County and Los Angeles County). Finally, estimates of risks above background PM concentrations are judged to be more relevant to policy decisions about the NAAQS than estimates that include risks potentially attributable to uncontrollable background PM concentrations.

The following sections summarize the planned scope of the risk analyses and key components of the risk model. A separate draft "Scoping Plan" (EPA, 2001c) is also available which provides a more detailed discussion. EPA plans to include and discuss the results from the risk analyses in the next draft of this Staff Paper.

4.2.1 Overview of Components of the Risk Model

In order to estimate the incidence of a particular health effect associated with "as is" conditions in a specific county attributable to ambient $PM_{2.5}$ or $PM_{10-2.5}$ exposures and the change in incidence of the health effect in that county corresponding to a given change in $PM_{2.5}$ and $PM_{10-2.5}$ levels resulting from just attaining a specified set of $PM_{2.5}$ and $PM_{10-2.5}$ standards, the following three elements are required:

- air quality information including: (1) "as is" air quality data for $PM_{2.5}$ and $PM_{10-2.5}$ from population-oriented monitors for the selected county, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations appropriate for that location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality estimated to occur when the county attains a given set of standards.

- relative-risk basedconcentration-response functions which provide an estimate of the relationship between the health endpoints of interest and ambient $PM_{2.5}$ and $PM_{10-2.5}$ concentrations.

- baseline health effects incidence or incidence rates which provide an estimate of the incidence or incidence rate of health effects corresponding to "as is" $PM_{2.5}$ and $PM_{10-2.5}$ levels.

Figure 4-1 provides a broad schematic depicting the role of these components in the risk analyses. Those points where EPA proposes to conduct analyses of alternative assumptions, procedures, or data are indicated by a circle with S_x in it. A fuller description of the type of sensitivity analyses planned is included in Table 4-1.

Most epidemiological studies estimating relationships between PM and health effects assume an exponential concentration-response function.[1] In this model,

$$y = B\, e^{\beta x} \quad , \qquad \text{(Equation 4-1)}$$

where x is the ambient PM level, y is the incidence of the health endpoint of interest at PM level x, β is the coefficient of ambient PM concentration, and B is the incidence at x=0, i.e., when there is no ambient PM. The change in health effects incidence from the baseline incidence, y (the incidence at "as is" PM concentration, x) to y_0 (the incidence at PM concentration x_0, attaining the alternative standards) corresponding to a given change in ambient PM levels, $\Delta x = x_0 - x$, is then

$$\Delta y = y[e^{\beta \Delta x} - 1] \qquad \text{(Equation 4-2)}$$

or, alternatively,

$$\Delta y = y(RR_{\Delta x} - 1) \qquad \text{(Equation 4-3)}$$

where $RR_{\Delta x}$ is the relative risk associated with the change in ambient PM levels, Δx.

[1]For some studies on respiratory hospital admissions used in the risk analysis a linear concentration-response function was estimated.

Figure 1. Major Components of Particulate Matter Health Risk Analysis

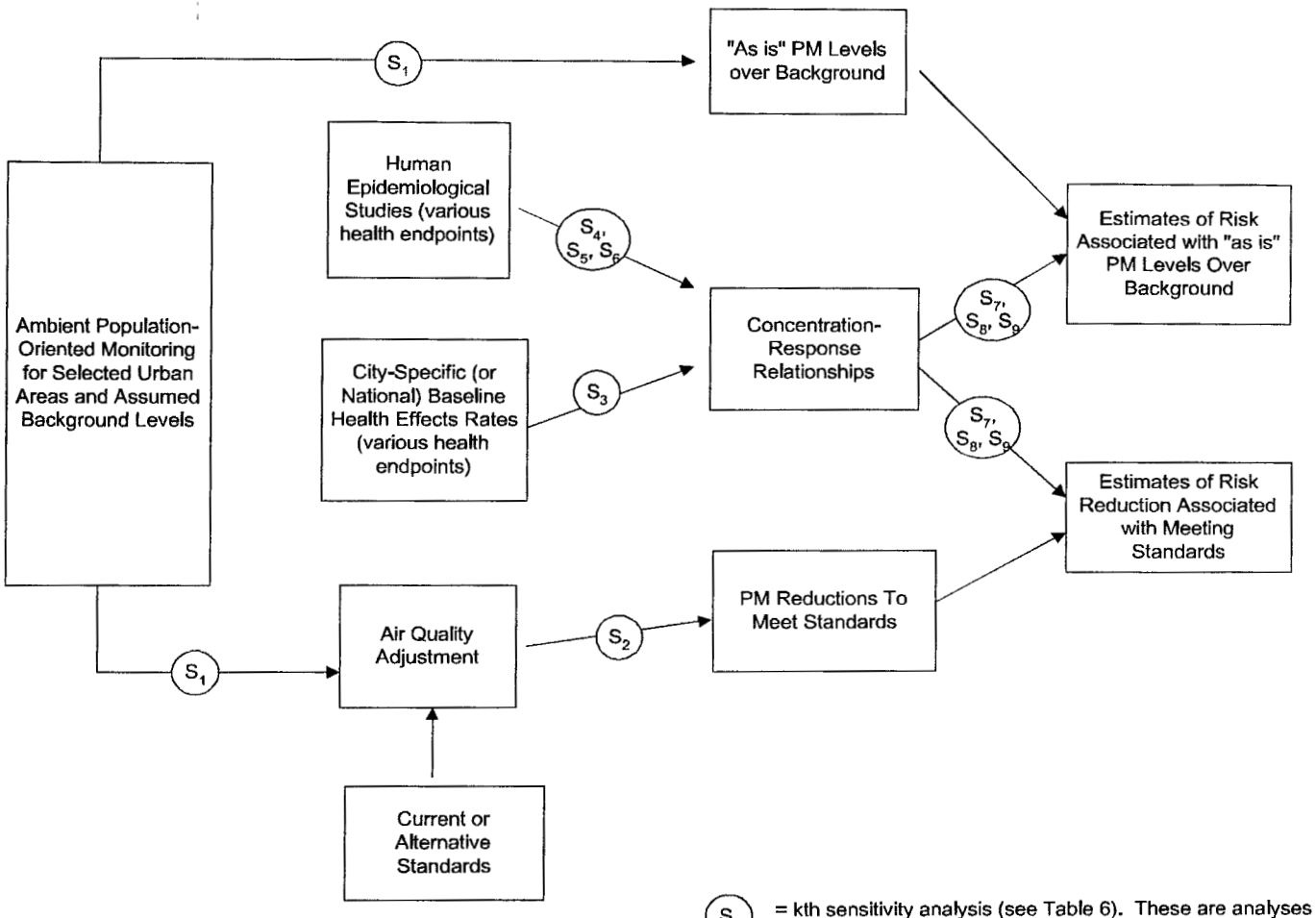

Table 4-1. Planned Sensitivity Analyses

Analysis Number (Figure 1)	Component of the Risk Analysis	Sensitivity Analysis or Comparison
1	Air Quality	A sensitivity analysis of the effect of different assumptions about background PM levels
2	Air Quality	A sensitivity analysis of the effect of different air quality adjustment procedures on the estimated risk reductions resulting from just meeting alternative 24-hr and annual standards
3	Baseline Incidence	A comparison of using more aggregate incidence data (national, state, etc) versus county-specific information in the county with the best local incidence data
4	Concentration-Response	A comparison or sensitivity analysis of methods of combining averaging times of from 1 to 5 days in the short-term mortality and hospital admissions studies
5	Concentration-Response	A sensitivity analysis or comparison of the effects of including or excluding individual studies from pooled functions to show the sensitivity of the function to inclusion of specific studies
6	Concentration-Response	A comparison or sensitivity analysis of the impact on mortality associated with long-term exposure of different assumptions about the role of historical air quality concentrations in contributing to the reported effects.
7	Concentration-Response	A sensitivity analysis comparing the risks estimated by using concentration-response functions derived for the specific county in question versus pooled functions for endpoints
8	Concentration-Response	A sensitivity analysis using concentration-response functions for PM from multi-pollutant regressions with co-pollutants versus single pollutant regressions
9	Concentration-Response	A sensitivity analysis assuming alternative minimum concentration levels for the occurrence of PM response at concentrations above those for background

Estimates of risk (i.e., incidences or incidence rates of health effects attributable to $PM_{2.5}$ or $PM_{10-2.5}$) will be quantified for $PM_{2.5}$ and $PM_{10-2.5}$ concentrations above background except for those studies in which the background concentration was not within the range of observable $PM_{2.5}$ or $PM_{10-2.5}$ concentrations used for the study (e.g., the prospective cohort mortality studies). For studies that do not evaluate risk at background levels, the effects will be quantified only down to the lowest concentrations observed in the study. Each of these key components is discussed below, highlighting those points at which judgments have been made that will determine the nature and scope of the risk analysis.

4.2.2 Air Quality Considerations

The air quality information required to conduct the PM risk analyses includes: (1) "as is" air quality data for both $PM_{2.5}$ and $PM_{10-2.5}$ from population-oriented monitors for the selected cities, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations appropriate to each location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality change estimated to occur when each location attains the current suite of $PM_{2.5}$ standards (as well as any potential alternative $PM_{2.5}$ standards identified as part of this review) or alternative $PM_{10-2.5}$ standards. Table 4-2 provides a summary of the $PM_{2.5}$ and $PM_{10-2.5}$ air quality data for the areas under consideration for inclusion in the risk analyses. The $PM_{10-2.5}$ observations are based on subtracting $PM_{2.5}$ concentrations from the PM_{10} concentration at a co-located monitoring site. Additional discussion of the available PM air quality data for these three locations is presented in the draft Scoping Plan (EPA, 2001c).

Table 4-2. Summary of PM Air Quality Data for Areas to Be Examined in PM Risk Analyses

Area	Population (millions)	Year	Number (%) of Days on Which Air Quality Data are Available		$PM_{2.5}$[b]		$PM_{10-2.5}$[b]	
			$PM_{2.5}$	$PM_{10-2.5}$	Annual Avg. ($\mu g/m^3$)	98th percentile 24-hr Avg.[a] ($\mu g/m^3$)	Annual Avg. ($\mu g/m3$)	98th Percentile 24-hr Avg.[b] ($\mu g/m^3$)
Philadelphia County, PA	1.4	1999	276 (75.6)	-	14.8	35.9	-	-
Los Angeles County, CA[c]	3.8	1998/1999	197 (54.0)	130 (35.6)	24.2	59.5	26.2	54
Salt Lake County, UT	0.85	1999	315 (86.0)	285 (78.0)	9.9	47	15.8	44

[a]The values shown in this column are the 98th percentile values at the "composite monitors" in Philadelphia and Los Angeles. The actual risk analyses will be based on the current form of the standard which requires the 98th percentile value at each monitor not exceed the standard.

[b]The value shown for Los Angeles is the 98th percentile value at the "composite monitor", while the 98th percentile value for Salt Lake County is the 98th percentile value at a specific monitor.

[c]The information in this row is for Southeast Los Angeles County which makes up a little over a third of Los Angeles County.

Background PM concentrations proposed to be used in the risk analyses are defined in Chapter 2 of this Staff Paper as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic emissions of PM and its precursors in North America. For the proposed risk analyses, an estimate of the annual average background level is desired, rather than a daily average (e.g., the maximum 24-hour level), since accumulated risks will be aggregated for each day throughout the year. The staff have chosen to use the midpoint of the appropriate ranges of annual average estimates for PM background presented in Chapter 2 for the base case risk estimates (i.e., eastern values will be used for Philadelphia County and western values will be used for Los Angeles and Salt Lake Counties).

- For $PM_{2.5}$: 2 to 5 $\mu g/m^3$ for Philadelphia and 1 to 4 $\mu g/m^3$ for Los Angeles and Salt Lake Counties

- For $PM_{10-2.5}$: 3 to 4 $\mu g/m^3$ for Los Angeles and Salt Lake Counties

Sensitivity analyses will be done using the appropriate lower and upper ends of the above ranges to characterize the impact of this model input choice on the risk estimates. OAQPS also recognizes that the estimated ranges for regional background levels of $PM_{10-2.5}$ due to natural sources and transport from outside of North America are more uncertain than the estimates for $PM_{2.5}$.

To estimate the health risks associated with just attaining the current $PM_{2.5}$ standards and alternative $PM_{10-2.5}$ standards, it is necessary to estimate PM concentrations that would occur under each specified standard (or sets of standards). When assessing the risks associated with long-term exposures, using epidemiological studies that use an annual average concentration, the annual mean is simply set equal to the standard level. In contrast, when assessing the risks associated with short-term exposures using epidemiological studies which consider daily average concentrations, the distribution of 24-hour values that would occur upon just attaining a given 24-hour PM standard has to be simulated. While there are many different methods of reducing daily PM levels, prior analyses conducted during the last NAAQS review found that PM levels have in general historically decreased in a proportional manner (i.e., concentrations at different points in the distribution of 24-hour PM values have decreased by approximately the same percentage) (Abt Associates, 1996b). Therefore, attainment of the current $PM_{2.5}$ daily standard and alternative daily $PM_{10-2.5}$ standards will be simulated by adjusting the "as is" air quality data using a proportional rollback approach (i.e., concentrations across the distribution are reduced by the same percentage) for concentrations exceeding the estimated background level. Sensitivity analyses will be conducted to examine alternative air quality adjustment procedures (e.g., a method that reduces the top 10% of daily PM concentrations more than the lower 90%).

4.2.3 Estimating Concentration-Response Functions

The second key component in the risk model is the set of concentration-response functions which provide estimates of the relationship between each health endpoint of interest and ambient

PM concentrations. The staff has selected the most significant health effect endpoints for which the weight of the evidence is supportive of an effect occurring. In cases where all of the available studies failed to find a statistically significant relationship, the effect endpoint was excluded. In situations where there is a mixture of statistically significant and non-significant findings for a given health effect endpoint and PM indicator (e.g., hospital admisisons for COPD patients and $PM_{2.5}$), staff also considered evidence from available PM_{10} studies in making a judgment on whether effects are likely related to PM.

The health endpoints that are proposed to be included in the $PM_{2.5}$ analyses include mortality (due to short- and long-term exposure), hospital admissions, emergency room visits, and respiratory illnesses and/or symptoms not requiring hospitalization. (Lung function studies will not be included.) Inclusion of a health endpoint in the analysis will be based on the weight of the evidence overall. Once it has been determined that a health endpoint will be included in the analysis, inclusion of a study on that health endpoint will not be based on the existence of a statistically significant result. That is, consistent with the approach taken in the prior PM risk analyses, no credible study on an included health endpoint will be excluded from the analysis on the basis of lack of statistically significant findings.

For the potential $PM_{10-2.5}$ risk analyses, EPA is considering including increased respiratory-related hospital admissions and increased respiratory symptoms as health endpoints. As discussed in Chapter 3 of this Staff Paper, these are the two health effect categories with the strongest evidence for effects being associated with $PM_{10-2.5}$ exposure. While there is evidence for other effects being associated with $PM_{10-2.5}$, the staff believes that the evidence is insufficient to justify conducting a quantitative risk analysis for other health endpoints. These other effects are addressed qualitatively in Chapter 3 of this Staff Paper.

Since the 1996 PM risk analyses were carried out, several new studies have investigated the relationship between PM and a health endpoint (e.g., short-term exposure mortality) in multiple cities using consistent methodological approaches in all locations examined. As noted in the draft CD (see, in particular, CD, Section 9.6.2.1.2), such multi-location studies are preferable, all else equal, to meta-analyses (i.e., pooling) of the results of multiple independent single-location studies carried out in different locations. The primary advantage of such multi-location studies is

the consistency in methodology used in all locations, eliminating the possibility that inter-locational differences might be due to differences in study design. In addition, multi-location studies are not subject to the omission of negative results due to publication bias that could affect a meta-analysis of the results of published single-location studies. Finally, any geographical variability in air pollution effects can be systematically evaluated in a multi-location study. For these reasons, such multi-location studies, if available, are preferred to meta-analyses of independent single-location studies.

Consistent with the approach taken in the prior PM risk analyses, if there is no multi-location study for a health endpoint, and if several single-location studies have been identified as appropriate for inclusion in the PM risk analyses, EPA proposes to combine the C-R functions from these studies to form a "pooled" estimate of the risk of that health effect attributable to $PM_{2.5}$ (or $PM_{10-2.5}$) and the risk reductions that would result from meeting current or alternative standards. The relationship between a pollutant and a health effect in a population may vary from one location to another due, for instance, to inter-locational differences in the composition of PM and/or the populations exposed. Pooling the estimates from several studies provides a central tendency estimate of the effect in any randomly selected location, as well as a characterization of the uncertainty about the effect in that location. The staff recognizes that caution is required in deciding which studies should be pooled for any given health endpoint and the draft Scoping Plan (EPA, 2001c) addresses in more detail the proposed principles that would be followed in selecting studies to be pooled.

In selecting studies to be considered for use in the PM risk analyses, the staff set forth several criteria, all of which have to be met to be included for consideration for the proposed risk analyses for this review. These include: (1) only studies cited in the draft CD tables (see CD, Tables 9-3, 9-4, and 9-6) or included in the prior 1996 risk analyses are included, (2) only studies conducted in the United States or Canada are included, (3) only studies that measured $PM_{2.5}$ (or $PM_{2.1}$) and/or $PM_{10-2.5}$ are included, and (4) only studies that are judged to be credible from a methodological standpoint are included. The staff recognizes that the draft CD is currently under review by both the CASAC and general public, and, thus, the final group of studies to be included in the analyses may change based on the review of the draft CD. Table 4-3 summarizes the

available epidemiological studies cited in the draft CD that may be useful in estimating total non-accidental and cause-specific mortality associated with short-term $PM_{2.5}$ exposures. Table 4-4 summarizes the available epidemiological studies cited in the draft CD that may be useful in estimating total and specific kinds of cardiovascular morbidity effects associated with $PM_{2.5}$ exposures. Table 4-5 summarizes the available epidemiological studies cited in the draft CD that may be useful in estimating total and specific kinds of respiratory morbidity effects associated with both $PM_{2.5}$ and $PM_{10-2.5}$ exposures.

In assessing or interpreting public health risk associated with exposure to PM, the form of the concentration-response function is an important component. The 1996 Criteria Document (EPA, 1996a) evaluated evidence from epidemiological studies regarding both functional form and whether a threshold for effects could be identified; this evaluation raised some key questions, but there was not sufficient evidence to draw conclusions (EPA, 1996a, Section 13.6.5).

Among the new epidemiological analyses are several studies that use different modeling methods to investigate potential threshold levels and concentration-response forms. As summarized in the draft CD, two of these studies presented no evidence of the existence of a threshold for associations between PM and acute mortality. Cakmak et al. (1999) tested different methods for detecting the presence of a threshold for the PM-mortality relationship, using Toronto pollution and mortality data. The authors concluded that "if threshold exists, it is highly unlikely that standard statistical analysis can detect it." (CD, p. 6-246). Similarly, Schwartz and Zanobetti (2000) used simulation methods with air quality data from 10 U.S. cities to investigate the presence of a threshold. No evidence was found for the existence of a threshold in the association between PM_{10} and short-term exposure mortality (CD, pp. 6-246, 247).

In addition, using data from 20 U.S. cities to analyze the PM_{10} and short-term exposure mortality relationship, roughly linear associations were found for total and cardiorespiratory mortality, consistent with the lack of a threshold.(CD, p. 6-238; Daniels et al., 2000). Some evidence for thresholds in the relationship between $PM_{2.5}$, but not $PM_{10-2.5}$, and mortality was found using data from Phoenix. Smith et al. (2000) found evidence suggesting a potential threshold level of 20-25 $\mu g/m^3$ for mortality associations with $PM_{2.5}$ but no evidence of a threshold in the relationship between $PM_{10-2.5}$ and mortality. The draft CD (CD, p. 6-247)

observes that the data set used in this analysis is small but the findings warrant further analysis. Overall, considering the results of these new studies, the draft CD concludes that "linear models without a threshold may well be appropriate for estimating the effects of PM_{10} on . . . mortality" (CD, p. 6-248), which is consistent with the conclusions of the previous Criteria Document (EPA, 1996a).

4.2.4 Baseline Health Effects Incidence Rates

The most common health risk model expresses the reduction in health risk (Δy) associated with a given reduction in PM concentrations (Δx) as a percentage of the baseline incidence (y). To accurately assess the impact of PM air quality on health risk in the selected cities, information on the baseline incidence of health effects (i.e., the incidence under "as is" air quality conditions) in each location is therefore needed. Where possible, county-specific incidences or incidence rates will be used. County-specific mortality incidences are available from the National Center for Health Statistics.

Table 4-3. Estimated Increased Mortality per Increments in 24-hr Concentrations of $PM_{2.5}$ from U.S. and Canadian Studies

Study Location (population studied and reference)*	RR (± CI) per 25 μg/m³ $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (μg/m³) (Min, Max) **
Total (nonaccidental) Mortality		
Six Cities (All ages) (Schwartz et al., 1996a)		
Portage, WI	*1.030 (0.993, 1.071)*	*11.2 (± 7.8)*
Topeka, KS	*1.020 (0.951, 1.092)*	*12.2 (± 7.4)*
Boston, MA	*1.056 (1.038, 1.074)*	*15.7 (± 9.2)*
St. Louis, MO	*1.028 (1.010, 1.043)*	*18.7 (± 10.5)*
Kingston/Knoxville, TN	*1.035 (1.005, 1.066)*	*20.8 (± 9.6)*
Steubenville, OH	*1.025 (0.998, 1.053)*	*29.6 (± 21.9)*
Overall Six-City results	*1.038 (1.028, 1.048)*	*median 14.7*
Overall Six-City results (Age 65+)	*1.043 (1.03, 1.056)*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.031 (0.004, 1.069)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.4 (-0.1, 2.9)	22 (4, 86)
Montreal, Canada (Goldberg et al., 2000) (All ages) (Age 65+)	 1.029 (0.99, 1.06) 1.033 (0.98, 1.069)	 3.3 (0, 30)
3 New Jersey Cities: Newark Camden Elizabeth (All ages) (Tsai et al., 2000)	 1.043 (1.028, 1.059) 1.057 (1.001, 1.115) 1.018 (0.946, 1.095)	 42.1 (± 22.0) 39.9 (± 18.0) 37.1 (± 19.8)
Philadelphia, PA (All ages) (Lipfert et al., 2000)	1.042 (p<0.055)	17.3 (-0.6, 72.6)
Phoenix, AZ (All ages) (Mar et al., 2000)	1.060 (1.00, 1.154)	13.0 (0, 42)
Phoenix, AZ (Age 65+) (Smith et al., 2000)	(>25 μg/m³) 2.868 (1.126, 7.250) (<25 μg/m³) 0.779 (0.610, 0.995)	NR
Santa Clara County, CA (All ages) (Fairley, 1999)	1.085 (1.032, 1.138)	13 (2, 105)
8 Canadian Cities (All ages) (Burnett et al., 2000)	1.030 (1.011, 1.050)	13.3 (max 86)

Study Location (population studied and reference)*	RR (± CI) per 25 μg/m³ $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (μg/m³) (Min, Max) **
Cause-Specific Mortality		
Cardiorespiratory:		
3 New Jersey Cities:		
Newark	1.051 (1.031, 1.072)	42.1 (±22.0)
Camden	1.062 (1.006, 1.121)	39.9 (±18.0)
Elizabeth	1.023 (0.95, 1.101)	37.1 (± 19.8)
(All ages) (Tsai et al., 2000)		
Total Cardiovascular:		
Six Cities (same as above) (All ages) (Schwartz et al., 1996)	*1.053 (1.035, 1.071)*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.032 (0.977, 1.089)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.027 (1.004, 1.049)	22 (4, 86)
Montreal, Canada (All ages) (Goldberg et al, 2000)	1.034 (0.988, 1.081)	17.4 (2.2, 72.0)
Philadelphia, PA (7-county area) (All ages) (Lipfert et al., 2000)	1.043 (p<0.055)	17.3 (-0.6, 72.6)
Phoenix, AZ (All ages) (Mar et al., 2000)	1.187 (1.057, 1.332)	13.0 (0, 42)
Santa Clara County, CA (All ages) (Fairley, 1999)	1.07 (p>0.05)	13 (2, 105)
Total Respiratory:		
Six Cities (same as above) (All ages) (Schwartz et al., 1996)	*1.085 to 1.103*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.023 (0.897, 1.166)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.027 (0.966, 1.091)	22 (4, 86)
Montreal, Canada (Goldberg et al., 2000)		
All ages	1.119 (1.015, 1.234))	3.3 (0, 30)
Age 65+	1.131 (1.019, 1.255)	
Philadelphia, PA (7-county area) (All ages) (Lipfert et al., 2000)	1.022 (p>0.055)	17.3 (-.6, 72.6)
Santa Clara County, CA (All ages) (Fairley, 1999)	1.12 (p>0.05)	13 (2, 105)

* Studies included in the prior 1996 risk analyses are in italics; new studies are in plain text.
** Relative risk (95% confidence interval), except for Fairley (1999) and Lipfert et al. (2000) where insufficient data are available to calculate confidence intervals so p-value is given in parentheses.
*** Min/Max 24-h PM indicator level shown in parentheses unless otherwise noted.

Table 4-4. Estimated Cardiovascular Morbidity Effects per Increments in 24-hr Concentrations of $PM_{2.5}$ from U.S. and Canadian Studies

Health Effect and Study Location (population studied and reference)*	RR (± CI) per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean ($\mu g/m^3$) (Min, Max) **
Increased Hospitalization		
Cardiovascular:		
Los Angeles, CA (Age 65+) Los Angeles, CA (Age 20-64) (Moolgavkar et al., 2000)	(age 65+) 1.043 (1.025, 1.061) (age 20-64) 1.035 (1.018, 1.053)	median 22 (4, 86)
Toronto, Canada (All ages) (Burnett et al., 1997)	1.072 (0.994, 1.156)	16.8 (1, 66)
Heart Failure:		
Detroit, MI *** (Lippmann et al., 2000)	1.091 (1.023, 1.162)	18 (6, 86)
Increased emergency department visits		
St. John, Canada (All ages) (Stieb et al., 2000)	1.151 (0.998, 1.328)	Summer 1993 8.5 (max 53.2)

Table 4-5. Estimated Respiratory Morbidity Effects per Increments in 24-hr Concentrations of $PM_{2.5}$ and $PM_{10-2.5}$ from U.S. and Canadian Studies

Study Location (population studied and reference)*	RR (± CI) per 25 μg/m³ $PM_{2.5}$ Increase	RR (± CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (μg/m³) (Min, Max) **
Increased Admission to Hospital			
Total Respiratory:			
Toronto, Canada (All ages) (Burnett et al., 1997)	1.086 (1.034, 1.141)	1.127 (1.052, 1.207)	$PM_{2.5}$ 16.8 (1, 66) PM_{10} 28.1 (4, 102) $PM_{10-2.5}$ 11.6 (1, 56)
Toronto, Canada (Age >64 years) (Thurston et al., 1994)	*1.15 (1.02, 1.28)*		*$PM_{2.5}$ 18.6 (NR, 66)*
Pneumonia:			
Detroit, MI (Age >65 years) **(Lippmann et al., 2000)**	1.125 (1.037, 1.220)	1.119 (1.007, 1.244)	$PM_{2.5}$ 18 (6, 86) PM_{10} 31 (max 105) $PM_{10-2.5}$ 13 (4, 50)
Respiratory infections:			
Toronto, Canada (All ages) (Burnett et al., 1997)	1.108 (1.072, 1.145)	1.093 (1.046, 1.142)	$PM_{2.5}$ 18.0 (max 90) PM_{10} 30.2 (max 116) $PM_{10-2.5}$ 12.2 (max 68)
COPD:			
Detroit, MI (All ages)(Lippmann et al., 2000)	1.055 (0.953, 1.168)		18 (6, 86)
King County, WA (All ages) (Moolgavkar et al., 2000)	1.065 (1.3, 1.118)	---	$PM_{2.5}$ 18.1 (3, 96) PM_{10}
Los Angeles, CA (Age >65 years) **(Moolgavkar et al., 2000)**	1.051 (1.009, 1.094)	---	$PM_{2.5}$ median 224, 86) PM_{10} median 44 (7, 166)
Increased respiratory emergency department visits			
Montreal, Canada (Age 65+) (Delfino et al., 1997)	1.239 (1.049, 1.428)	---	summer 1993 $PM_{2.5}$ 12.2 (max 31) PM_{10} 21.7 (max 51)
St. John, Canada (All ages) (Stieb et al., 2000)	1.057 (1.006, 1.110)	---	summer 1993 $PM_{2.5}$ 8.5 (max 53.2) PM_{10} 14.0 (max 70.3)

Study Location (population studied and reference)*	RR (± CI) per 25 μg/m³ $PM_{2.5}$ Increase	RR (± CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (μg/m³) (Min, Max) **
Asthma:			
Increased Respiratory Symptoms			
Uniontown, PA (evening cough) (Neas et al., 1995)	*1.45 (1.07, 1.97)*		*24.5 (max 88.1)*
Southwest Virginia (Runny or Stuffy nose) (Zhang et al., 2000)		2.62 (1.16, 5.87)	$PM_{2.5}$ NR $PM_{10-2.5}$ NR
State College, PA Cough Cold (Neas et al., 1996)	 1.61 (1.21,2.17) 1.45 (1.29, 4.64)	 ---	PM_{21} 23.5 (max 85.8) $PM_{10-2.5}$ ---
Six Cities reanalysis : Cough Lower respiratory symptoms (Children grades 2-5) (Schwartz and Neas, 2000)		 1.77 (1.23, 2.54) 1.51 (0.94, 4.87)	 $PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Six Cities: *Cough* *Lower respiratory symptoms (LRS)* *(Children grades 2-5) (Schwartz et al., 1994)*	 *1.24 (1.00, 1.54)* *1.58 (1.18, 2.10)*		*18.0 (max 86.0)*

Table 4-6. Effect Estimates per Increments in Long-term Mean Levels of Fine Particle Indicators from U.S. and Canadian Studies

Type of Health Effect and Study Location (population studied and reference)	RR (± CI) per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	Range of City $PM_{2.5}$ Levels, Means ($\mu g/m^3$)
Increased total mortality in adults		
Six Cities Reanalysis (Age 25+) (Krewski et al., 2000)	1.39	11-30
ACS Study Reanalysis (Age 30+) (Krewski et al., 2000)	1.18	9-33
Increased cardiopulmonary mortality in adults		
Six Cities Reanalysis (Age 25+) (Krewski et al., 2000)	1.45	11-30
ACS Study Reanalysis (Age 30+) (Krewski et al., 2000)	1.31	9-33

For many of the morbidity endpoints, however, county-specific incidence rates are difficult to obtain. County-specific rates for hospital admissions are in the process of being obtained for Philadelphia, Los Angeles, and Salt Lake counties. For other morbidity endpoints, such as respiratory symptoms in children, incidence information aggregated at a higher level may be all that is available. The level of aggregation closest to county-specific will be used; however, for some morbidity endpoints, it may be necessary to estimate county-specific incidence using national-level incidence rates. For some health endpoints, there may be no information on incidence other than the information provided for the city in which the concentration-response function was estimated. A discussion will be presented of the rationale for the choice of incidence data used for each location. The lack of city- or county-specific incidence data will increase uncertainty concerning the estimates of risk for the specific cities selected for the risk analysis.

To the extent possible, a quantitative comparison will be provided to help assess the accuracy of using incidence rates at a higher level of aggregation (e.g., national incidence rates) by comparing these rates to city- or county-specific incidence rates where these are available.

4.2.5 Uncertainties in Risk Analyses and Plans for Conducting Sensitivity Analyses

There are considerable uncertainties in risk analyses for any air pollutant. These are compounded in the case of a pollutant such as PM (as opposed to, for example, O_3), given the diversity of composition in this generally defined pollutant. Among the major sources of uncertainty in the planned risk analyses are:

- The statistical uncertainty surrounding estimates of PM coefficients in concentration-response functions used in the analysis.

- The transferability of PM concentration-response functions from study locations to the locations selected for the risk analysis due to variations in PM composition across cities; the possible role of associated copollutants in influencing PM risk; and variations in the relation of ambient exposure to ambient monitoring in different locations. There is also uncertainty concerning the transferability of health functions to future PM aerosol mixes. In addition, cities may have different population sensitivity to PM effects (with some sensitive populations likely still to be defined).

- The air quality adjustment procedure that will be used to simulate just meeting alternative PM standards, and uncertainty about the extent to which reductions in PM will consist of reductions in fine versus coarse particles.

- Use of baseline health effects incidence information that is not specific to the county in question.

- Applying pooled concentration-response functions to represent the overall effect of particles on a particular health endpoint from studies in several locations.

- The impact of historical air quality on estimates of health risk from long-term PM exposures – the duration of time that a reduction in particle concentrations must be maintained in a given location in order to experience the predicted reduction in health risk and/or the possibility of lags between exposure and health effect.

- The effect of normalizing to different degrees the amounts of health risk experienced or reduced in different locations because of differences in the completeness of the air quality data sets.

- Estimated background concentrations for each location.

- The effect of measurement uncertainty in the original health studies used to develop the concentration-response relationships.

The uncertainties from some of these sources – in particular, the statistical uncertainty surrounding estimates of pollutant coefficients – can be characterized quantitatively. It will be possible, for example, to calculate confidence intervals around risk estimates based on the statistical uncertainty associated with the estimates of pollutant coefficients used in the risk analyses. These confidence intervals will express the range within which the true risks are likely to fall *if the statistical uncertainty surrounding pollutant coefficient estimates were the only uncertainty in the analyses*. There are, of course, several other uncertainties in the risk analyses, as noted above. If there were sufficient information to quantitatively characterize these sources of uncertainty, they could be included in a Monte Carlo analysis to produce confidence intervals that more accurately reflect all sources of uncertainty.

Uncertainties in the risk analysis are proposed to be handled in the following ways:

- Limitations and assumptions in the quantification process will be clearly stated and explained.

- For any endpoint for which only a single concentration-response function has been estimated, the uncertainty resulting from the statistical uncertainty associated with the estimate of the pollutant coefficient will be characterized by confidence intervals around the point estimate of risk. As noted above, such a confidence interval will express the range within which the true risk is likely to fall *if the statistical uncertainty surrounding the pollutant coefficient estimate were the only uncertainty in the analysis*. It will not, for example, reflect the uncertainty concerning whether the pollutant coefficients in the study location and the assessment location are the same.[2]

- For any endpoint for which a pooled function has been derived from two or more studies, a credible interval will be presented along with the point estimate of risk. Credible intervals will reflect not only the within-study statistical uncertainty, but the between-study variability in pollutant coefficients as well. These credible intervals will therefore, to some extent, also reflect the uncertainty associated with applying functions estimated in locations other than the assessment location.

[2] This is not an uncertainty, of course, if the concentration-response function has been estimated in the assessment location.

- Sensitivity analyses will be conducted to illustrate the effects of changing key default assumptions on the mean results of the assessment, and quantitative comparisons[3] presented to inform other analytic choices.

Possible additional or alternative approaches to characterizing uncertainty that are being considered include the following:

- To include in an overall assessment of uncertainty those sources of uncertainty that cannot readily be quantified, "integrated sensitivity analyses" may be presented. These analyses rely on staff judgment to assign probabilities to possible alternatives. For example, staff judgment would be used to assess the likelihood that each of several possible alternative assumptions is the correct one. This procedure allows sources of uncertainty that are otherwise not quantifiable to be included in a Monte Carlo analysis of overall sensitivity to various alternative values.

- Different sets of plausible assumptions that would result in "low end," "middle," and "high end" estimates of incidence could be identified, and the estimates resulting under each set of assumptions could be presented as alternatives.

4.3 $PM_{2.5}$ Risk Estimates for Philadelphia and Los Angeles Counties

The next draft of the Staff Paper will include presentation of base case risk estimates for "as is" air quality, air quality levels associated with just attaining the current $PM_{2.5}$ standards, and air quality associated with attaining any potential alternative $PM_{2.5}$ standards that are identified as part of this review. In addition, results of sensitivity analyses of individual uncertainties and assumptions as well as integrated uncertainty analyses examining the impact of several key uncertainties will be presented. This section will then conclude with key observations from the $PM_{2.5}$ risk analyses.

4.4 $PM_{10-2.5}$ Risk Estimates for Example Counties

If the Agency decides to conduct $PM_{10-2.5}$ risk analyses, this section will include base case risk estimates for as is air quality, air quality levels associated with just attaining the current $PM_{2.5}$ standards, and air quality associated with attaining any alternative $PM_{10-2.5}$ standards that are

[3]"Sensitivity analyses" refers to assessing the effects of uncertainty on some of the final risk estimates; "quantitative comparisons" refer to numerical comparisons (e.g. comparisons of monitor values) that are not carried that far.

identified as part of this review. In addition, results of sensitivity analyses of individual uncertainties and assumptions as well as integrated uncertainty analyses examining the impact of several key uncertainties will be presented. This section will then conclude with key observations from the $PM_{10-2.5}$ risk analyses.

REFERENCES

Most Chapter 4 references are available at the end of Chapter 3. References not listed at the end of Chapter 3 are listed here.

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5. CHARACTERIZATION OF PM-RELATED ENVIRONMENTAL EFFECTS

5.1 INTRODUCTION

This chapter summarizes key information relevant to assessing the environmental effects associated with ambient PM, alone and in combination with other pollutants commonly present in the ambient air, drawing upon the most relevant information contained in the draft CD and other significant reports referenced therein. The chapter is organized into a discussion of the effects on public welfare to be considered in this review of the secondary standards for PM. Specifically, this chapter addresses PM-related effects on visibility (Section 5.2), materials (Section 5.3), vegetation and ecosystems (Section 5.4), and solar radiation and global climate change (Section 5.5). For each category of PM-related effects, this preliminary draft chapter presents a brief summary of the relevant scientific information and a preliminary staff assessment of whether the available information is sufficient to be considered as the basis for secondary standards distinct from primary standards for PM. In addition, in assessing information on PM-related effects on solar radiation and global climate change, consideration is given to potential indirect impacts on human health and the environment that may be a consequence of radiative and climatic changes attributable to changes in ambient PM. Staff conclusions and recommendations related to secondary standards for PM will be incorporated into Chapter 6 of a subsequent draft of this Staff Paper.

It is important to note that the discussion of PM-related effects on visibility, vegetation and ecosystems, and solar radiation and global climate change in Chapter 4 of the draft CD builds upon and includes by reference extensive information from several other significant reviews of these areas. Most notably, these reports include the Recommendations of the Grand Canyon Visibility Transport Commission (1996), the National Research Council's *Protecting Visibility in National Parks and Wilderness Areas* (1993), reports of the National Acid Precipitation Assessment Program (1991), previous EPA Criteria Documents, including *Air Quality Criteria for Particulate Matter and Sulfur Oxides* (EPA, 1982) and *Air Quality Criteria for Oxides of Nitrogen* (EPA, 1993), and numerous U.S. and international assessments of stratospheric ozone depletion and global climate change carried out under U.S. Federal interagency programs (e.g.,

the U.S. Global Climate Change Research Program) and the World Meteorological Organization (WMO) and the United Nations Environment Programme (UNEP).

5.2 EFFECTS ON VISIBILITY

Visibility impairment has long been considered the "best understood and most easily measured effect of air pollution" (Council on Environmental Quality, 1978). It is caused by the scattering and absorption of light by particles and gases in the atmosphere. It is the most noticeable effect of fine particles present in the atmosphere. Air pollution degrades the visual appearance of distant objects to an observer, and reduces the range at which they can be distinguished from the background. Ambient particles affect the perceived color of distant objects depending upon particle size and composition, the scattering angle between the observer and illumination, the properties of the atmosphere, and the optical properties of the target being viewed.

This section discusses the role of ambient PM in the impairment of visibility, building upon the information present in the last Staff Paper (EPA, 1996b) and drawing upon the most relevant information contained in the draft CD and significant reports on the science of visibility referenced therein. In particular, this section includes new information on the following topics:

- Planned data analyses to characterize visibility impairment in urban and suburban areas based on 1999 visibility data from 60+ Automated Surface Observation System (ASOS) installations from around the country, and to explore the degree to which the ASOS data correlates with 1999 daily $PM_{2.5}$ measurements.

- An overview of existing and planned visibility programs, goals, and methods for the evaluation of visibility impairment as a basis for standard setting, in the U.S. and abroad, illustrating the significant value placed on efforts to improve visibility outside of national parks and wilderness areas.

- A pilot survey project conducted by EPA in November 2000 in Washington DC to elicit public input on the acceptability of varying levels of visual air quality in urban areas, and plans for conducting a broader survey using the methodology developed and refined as part of the pilot project, using new techniques for photographic representation of visibility impairment.

The presentation here organizes the available information on visibility impairment into elements related to the evaluation of current and alternative standards for PM. Beyond providing an overview of visibility impairment, this section summarizes: (1) the effects of PM on visibility (building upon information presented above in Section 2.9); (2) conditions in Class I and non-urban areas, as well as in urban areas; (3) information on the significance of visibility to public welfare; and (4) approaches to evaluating public perceptions of visibility impairment and judgments about the acceptability of varying degrees of impairment.

5.2.1 Overview of Visibility Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light (NRC, 1993; CD, 4-86). Visibility effects are manifested in two principal ways: (1) as local impairment (e.g., localized hazes and plumes); and (2) as regional haze. These distinctions are significant both to the ways in which visibility goals may be set and air quality management strategies may be devised.

Local-scale visibility degradation is commonly in the form of either a plume resulting from the emissions of a specific source or small group of sources, or it is in the form of a localized haze, such as an urban "brown cloud." Impairment caused by a specific source or small group of sources has been generally termed as "reasonably attributable" impairment. Plumes are comprised of smoke, dust, or colored gas that obscure the sky or horizon relatively near sources. Sources of locally visible plumes, such as the plume from an industrial facility or a burning field, are often easy to identify. "Reasonably attributable" impairment may include contributions to local hazes by individual sources or several identified sources. There have been a limited number of cases in which Federal land managers have certified the existence of visibility impairment in a Class I area (i.e., 156 national parks, wilderness areas, and international parks identified for visibility protection in section 162(a) of the Clean Air Act) that is considered "reasonably attributable" to a particular source.[1]

[1]Two of the most notable cases leading to emissions controls involved the Navajo Generating Station in Arizona and the Mohave power plant in Nevada, for which it was found that sulfur dioxide emissions were contributing to visibility impairment in Grand Canyon National Park.

A localized or layered haze often results from emissions from many sources located across an urban or metropolitan area. This type of impairment may be seen as a band or layer of discoloration appearing well above the terrain. A common manifestation of this type of visibility impairment is the "brown cloud" situation experienced in some cities particularly in the winter months, when cooler temperatures limit vertical mixing of the atmosphere. Urban visibility impairment often results from the combined effect of stationary, mobile, and area source emissions, and complex local meteorological conditions may contribute to such impairment as well. The long-range transport of emissions from sources outside the urban area may also contribute to urban haze levels. A number of studies have been conducted in the past in cities like Denver, Dallas, and Seattle to characterize urban visibility problems.

The second type of impairment, regional haze, results from pollutant emissions from a multitude of sources located across a broad geographic region. It impairs visibility in every direction over a large area, in some cases over multi-state regions. Regional haze masks objects on the horizon and reduces the contrast of nearby objects. The formation, extent, and intensity of regional haze is a function of meteorological and chemical processes, which sometimes cause fine particle loadings to remain suspended in the atmosphere for several days and to be transported hundreds of kilometers from their sources (NRC, 1993). It is this second type of visibility degradation that is principally responsible for impairment in national parks and wilderness areas across the country (NRC, 1993). Visibility in urban areas at times may be dominated by local sources, but often may be significantly affected by long-range transport of haze due to the multi-day residence times of fine particles in the atmosphere. Fine particles transported from urban areas in turn may be significant contributors to regional-scale impairment in Class I and other rural areas.

5.2.2 Effects of PM on Visibility

The efficiency at which a unit mass of particles causes visibility impairment depends on a number of factors, including particle size, composition, and humidity. These basic concepts are discussed above in Section 2.9.1. Building on this information, this section discusses common

measures of visibility impairment, estimated natural visibility conditions, and other important factors in the relationship between PM and visibility impairment.

5.2.2.1 Measures of Visibility Impairment

Several atmospheric optical indices and approaches can be used for characterizing visibility impairment. As summarized below and discussed in more detail in the draft CD, there are several indicators that could be used in regulating air quality for visibility protection, including: (1) human observation of visual range; (2) light extinction (and related parameters of visual range and deciview); (3) light scattering by particles; and (4) fine particle mass concentration (CD, page 4-94).

Human Observation. For many decades, the National Weather Service has recorded hourly visibility at major airports based on human observations of distant targets. This approach has provided a historical record of visibility across the U.S. and has allowed a general interpretation of regional visibility trends. Airport visibility monitoring has been automated in recent years, however, through deployment of the Automated Surface Observing System (ASOS) at more than 900 airports across the country (discussed below in Section 5.2.5). While human observations have been very effective for the purposes of air safety, these data are not as well correlated to air quality levels as data obtained from other automated monitoring methods.

Light Extinction and Related Measures. The light extinction coefficient has been widely used in the U.S. for many years as a metric to describe the effect of pollutant concentrations on visibility. It can be defined as the fraction of light lost or redirected per unit distance through interactions with gases and suspended particles in the atmosphere. The light extinction coefficient represents the summation of light scattering and light absorption due to particles and gases in the atmosphere. Both anthropogenic and non-anthropogenic sources contribute to light extinction. The light extinction coefficient (σ_{ext}) is represented by the following equation (CD, 4-89):

$$\sigma_{ext} = \sigma_{sg} + \sigma_{ag} + \sigma_{sp} + \sigma_{ap}$$

where σ_{sg} = light scattering by gases (also known as Rayleigh scattering)

σ_{ag} = light absorption by gases

σ_{sp} = light scattering by particles

σ_{ap} = light absorption by particles.

Light extinction is commonly expressed in terms of inverse kilometers (km^{-1}) or inverse megameters (Mm^{-1}), where increasing values indicate increasing impairment.

Total light extinction can be measured directly by a transmissometer or it can be calculated from ambient pollutant concentrations. Transmissometers measure the light transmitted through the atmosphere over a distance of 1 to 15 km. The light transmitted between the light source (transmitter) and the light-monitoring component (receiver) is converted to the path-averaged light extinction coefficient. Transmissometers operate continuously, and data is often reported in terms of hourly averages.

Direct relationships exist between measured ambient pollutant concentrations and their contributions to the extinction coefficient. The contribution of each aerosol constituent to total light extinction is derived by multiplying the aerosol concentration by the extinction efficiency for that aerosol constituent. Extinction efficiencies vary by type of aerosol constituent and have been obtained through empirical studies. For certain aerosol constituents, extinction efficiencies increase significantly with increases in relative humidity.

In addition to the optical effects of atmospheric constituents as characterized by the extinction coefficient, lighting conditions and scene characteristics play an important role in determining how well we see objects at a distance. Some of the conditions that influence visibility include whether a scene is viewed towards the sun or away from it, whether the scene is shaded or not, and the color and reflectance of the scene (NAPAP, 1991). For example, a mountain peak in bright sun can be seen from a much greater distance when covered with snow than when it is not.

One's ability to clearly see an object is degraded both by the reduction of image forming light from the object caused by scattering and absorption, and by the addition of non-image forming light that is scattered into the viewer's sight path. This non-image forming light is called path radiance (EPA, 1996a, p. 8-23). A common example of this effect is our inability to see stars in the daytime due to the brightness of the sky caused by Rayleigh scattering. At night, when the sunlight is not being scattered, the stars are readily seen. This same effect causes a haze to appear

bright when looking at scenes that are generally towards the direction of the sun and dark when looking away from the sun.

Though these non-air quality related influences on visibility can sometimes be significant, they cannot be accounted for in any practical sense in formulation of national or regional measures to minimize haze. Lighting conditions change continuously as the sun moves across the sky and as cloud conditions vary. Non-air quality influences on visibility also change when a viewer of a scene simply turns their head. Regardless of the lighting and scene conditions, however, sufficient changes in ambient concentrations of PM will lead to changes in visibility (and the extinction coefficient). The extinction coefficient integrates the effects of aerosols on visibility, yet is not dependent on scene-specific characteristics. It measures the changes in visibility linked to emissions of gases and particles that are subject to some form of human control and potential regulation, and therefore can be useful in comparing visibility impact potential of various air quality management strategies over time and space (NAPAP, 1991).

By apportioning the extinction coefficient to different aerosol constituents, one can estimate changes in visibility due to changes in constituent concentrations (Pitchford and Malm, 1994). The National Research Council's 1993 report *Protecting Visibility in National Parks and Wilderness Areas* states that "[p]rogress toward the visibility goal should be measured in terms of the extinction coefficient, and extinction measurements should be routine and systematic." Thus, it is reasonable to use the change in the light extinction coefficient, determined in multiple ways, as the primary indicator of changes in visibility for regulatory purposes.

Visual range is a measure of visibility that is inversely related to the extinction coefficient. Visual range can be defined as the maximum distance at which one can identify a black object against the horizon sky. The colors and fine detail of many objects will be lost at a distance much less than the visual range, however. Visual range has been widely used in air transportation and military operations in addition to its use in characterizing air quality. Conversion from the extinction coefficient to visual range can be made with the following equation (NAPAP, 1991):

$$\text{Visual Range } (km^{-1}) = 3.91/\sigma_{ext}$$

Another important visibility metric is the deciview, a unitless metric which describes changes in uniform atmospheric extinction that can be perceived by a human observer. It is designed to be linear with respect to perceived visual changes over its entire range in a way that is analogous to the decibel scale for sound (Pitchford and Malm, 1994). Neither visual range nor the extinction coefficient has this property. For example, a 5 km change in visual range or 0.01 km^{-1} change in extinction coefficient can result in a change that is either imperceptible or very apparent depending on baseline visibility conditions. The deciview metric allows one to more effectively express perceptible changes in visibility, regardless of baseline conditions. A one deciview change is a small but perceptible scenic change under many conditions, approximately equal to a 10% change in the extinction coefficient. The deciview metric also may be useful in defining goals for perceptible changes in visibility conditions under future regulatory programs. Deciview can be calculated from the light extinction coefficient (σ_{ext}) by the equation:

$$dv = 10 \log_{10}(\sigma_{ext}/10\ Mm^{-1})$$

Figure 5-1 graphically illustrates the relationships among light extinction, visual range, and deciview.

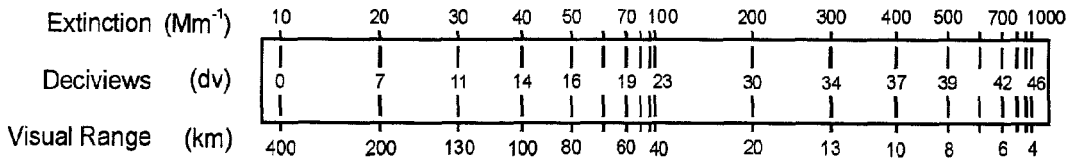

Figure 5-1. Relationship Between Light Extinction, Deciview, and Visual Range.

Light Scattering Coefficient. Across the U.S., light scattering is typically a much larger contributor to total light extinction than light absorption. Of the main categories of particles, only elemental carbon is a key contributor to light absorption and commonly represents only 5-10% of total light extinction (Malm et al., 2000). Light scattering data taken by a nephelometer can be

correlated fairly well with total light extinction measurements using certain assumptions for light absorption. Nephelometers measure the scattering of light by particles contained in a small volume of air, and thus provide a point measurement of scattering.

Fine Particle Mass Concentration. Fine particle (e.g., $PM_{2.5}$) mass concentrations can be used as a general surrogate for visibility impairment. However, as described in many reviews of the science of visibility, the different constituents of $PM_{2.5}$ have variable effects on visibility impairment. For example, crustal material in general accounts for less light scattering per unit mass than other constituents, and sulfates and nitrates contribute greater amounts of light scattering as relative humidity levels exceed 70%. Thus, while higher $PM_{2.5}$ mass concentrations generally indicate higher levels of visibility impairment, it is not as precise a metric as the light extinction coefficient. By using historic averages or regional estimates of the component-specific percentage of total mass, however, one can develop reasonable estimates of light extinction from PM mass concentrations.

5.2.2.2 Rayleigh Scattering and Natural Background Conditions

Rayleigh scattering represents the degree of natural light scattering found in a particle-free atmosphere, caused by the gas molecules that make up "blue sky" (e.g., N_2, O_2). It accounts for a relatively constant level of light extinction nationally, between 10 to 12 Mm^{-1} (NAPAP, 1991; EPA, 1979). The concept of Rayleigh scattering can be used to establish a theoretical maximum horizontal visual range in the earth's atmosphere. At sea level, this maximum visual range is approximately 330 kilometers. Since certain meteorological circumstances can reduce pollution that can result in visibility conditions that are close to "Rayleigh," it is analogous to a baseline or boundary condition against which other extinction components can be compared.

Light extinction caused by PM from natural sources can vary significantly from day to day and location to location due to natural events such as wildfire, dust storms, and volcanic eruptions. It is useful to consider estimates of natural background concentrations of PM on an annual average basis, however, when evaluating the relative contributions of anthropogenic (man-made) and non-anthropogenic sources to total light extinction.

As discussed in Chapter 2, for the purpose of this document, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of

anthropogenic emissions of primary PM and precursor emissions of VOC, NO_x, SO_2, and NH_3 in North America. Table 2-4 describes the range for annual average regional background $PM_{2.5}$ mass in the eastern U.S. as 2 to 5 $\mu g/m^3$, and in the western U.S. as 1 to 4 $\mu g/m^3$. For PM_{10}, the estimated annual average background concentrations range from 5 to 11 $\mu g/m^3$ in the eastern U.S., and 4 to 8 $\mu g/m^3$ in the western U.S.

The NAPAP report provides estimates of extinction contributions from Rayleigh scattering plus background levels of fine and coarse particles. In the absence of anthropogenic emissions of visibility-impairing particles, these estimates are 26 ± 7 Mm^{-1} in the East, and 17 ± 2.5 Mm^{-1} in the West. These equate to a naturally-occurring visual range in the East of 150 ± 45 km, and 230 ± 40 km in the West. Excluding light extinction due to Rayleigh scatter, annual average background levels of fine and coarse particles are estimated to account for 14 Mm^{-1} in the East and about 6 Mm^{-1} in the West. Major contributors that reduce visibility from the Rayleigh maximum to the ranges noted above are naturally-occurring organics, suspended dust (including coarse particles), and water. In these ranges of fine particle concentrations, small changes have a large effect on total extinction. Thus, higher levels of background fine particles and associated humidity in the East result in a fairly significant difference between naturally-occurring visual range in the rural East and West.

5.2.2.3 Contribution of PM to Visibility Conditions

On an annual average basis, the concentrations of background fine particles are generally small when compared with concentrations of fine particles from anthropogenic sources (NRC, 1993). The same relationship holds true when one compares annual average light extinction due to background fine particles with light extinction due to background plus anthropogenic sources. Table VIII-4 in the 1996 Staff Paper (EPA 1996b, p. VIII-10b) makes this comparison for several locations across the country by using background estimates from Table VIII-2 (EPA 1996b, p. VIII-6a) and light extinction values derived from monitored data from the IMPROVE network. These data indicate that anthropogenic emissions make a significant contribution to average light extinction in most parts of the country, as compared to the contribution from background fine particle levels. Man-made contributions account for about one-third of the average extinction coefficient in the rural West and more than 80% in the rural East (NAPAP, 1991).

It is important to note that even in those areas with relatively low concentrations of anthropogenic fine particles, such as the Colorado plateau, small increases in anthropogenic fine particle concentrations can lead to significant decreases in visual range. This is one reason why Class I areas have been given special consideration under the Clean Air Act. This relationship is illustrated by Figure VIII-9 in the 1996 Staff Paper (EPA, 1996b, p. VIII-10c) which relates changes in fine particle concentrations to changes in visibility (represented by the deciview metric). The graph shows that the visibility in an area with lower concentrations of air pollutants (such as many western Class I areas) will be more sensitive to a given increase in fine particle concentration than a more polluted atmosphere will be. Conversely, to achieve a given amount of visibility improvement, a larger reduction in fine particle concentration is required in areas with higher existing concentrations, such as the East, than would be required in areas with lower concentrations.

This relationship also illustrates the relative importance of the overall extinction efficiency of the pollutant mix at particular locations. At a given ambient concentration, areas having higher average extinction efficiencies due to the mix of pollutants would have higher levels of impairment (EPA, 1996b, p. VIII-10c, Figure VIII-9). In the East, the combination of higher humidity levels and a greater percentage of sulfate as compared to the West causes the average extinction efficiency for fine particles to be almost twice that for sites on the Colorado Plateau.

5.2.3 Visibility Conditions in Class I and Non-Urban Areas

5.2.3.1 IMPROVE Visibility Monitoring Network

In conjunction with the National Park Service, other Federal land managers, and State organizations, EPA has supported monitoring in national parks and wilderness areas since 1988. The network was originally established at 30 sites, but it has now been expanded to 110 of the 156 mandatory Federal Class I areas across the country. This long-term visibility monitoring network is known as IMPROVE (Interagency Monitoring of PROtected Visual Environments. The following discussion briefly describes the IMPROVE protocol and provides rationale supporting use of the light extinction coefficient, derived from both direct optical measurements

and measurements of aerosol constituents, for purposes of implementing air quality management programs to improve visibility.

IMPROVE provides direct measurement of fine particles and precursors that contribute to visibility impairment. The IMPROVE network employs aerosol, optical, and scene measurements. Aerosol measurements are taken for PM_{10} and $PM_{2.5}$ mass, and for key constituents of $PM_{2.5}$, such as sulfate, nitrate, organic and elemental carbon, soil dust, and several other elements. Measurements for specific aerosol constituents are used to calculate "reconstructed" aerosol light extinction by multiplying the mass for each constituent by its empirically-derived scattering and/or absorption efficiency. Knowledge of the main constituents of a site's light extinction "budget" is critical for source apportionment and control strategy development. Optical measurements are used to directly measure light extinction or its components. Such measurements are taken principally with either a transmissometer, which measures total light extinction, or a nephelometer, which measures particle scattering (the largest human-caused component of total extinction). Scene characteristics are recorded 3 times daily with 35 millimeter photography and are used to determine the quality of visibility conditions (such as effects on color and contrast) associated with specific levels of light extinction as measured under both direct and aerosol-related methods. Because light extinction levels are derived in two ways under the IMPROVE protocol, this overall approach provides a cross-check in establishing current visibility conditions and trends and in determining how proposed changes in atmospheric constituents would affect future visibility conditions.

5.2.3.2 Current Conditions Based on IMPROVE Data

Annual average visibility conditions (i.e., total light extinction due to anthropogenic and non-anthropogenic sources) vary regionally across the U.S. The rural East generally has higher levels of impairment than remote sites in the West, with the exception of the San Gorgonio Wilderness (CA), Point Reyes National Seashore (CA), and Mount Rainier National Park (WA), which have annual average levels comparable to certain sites in the Northeast. Higher averages in the East are due to generally higher concentrations of anthropogenic fine particles and higher average relative humidity levels. Visibility conditions also vary significantly by season of the year. With the exception of remote sites in the northwestern U.S., visibility is typically worse in the

summer months. This is particularly true in the Appalachian region, where average extinction in the summer exceeds the annual average by 40% (Sisler et al., 1996).

At this time, the 1996 Staff Paper serves as a general reference for understanding rural visibility conditions based on IMPROVE data. The next draft of this Staff Paper will include updated visibility trends and information on current conditions based on the latest available data.

5.2.4 Urban Visibility Conditions

For many years, urban visibility has been characterized using data describing airport visibility conditions. Until the mid-1990's, airport visibility was typically reported on an hourly basis by human observers. An extensive database of these assessments has been maintained and analyzed to characterize visibility trends from the late-1940's to mid-1990's (Schichtel et al., 2000).

As noted earlier, visibility impairment has been studied in several major cities in the past decades because of concerns about fine particles and their potentially significant impacts (e.g., health-related and aesthetic) on the residents of large metropolitan areas (e.g,. Middleton, 1993). Urban areas generally have higher loadings of fine particles and higher visibility impairment levels than monitored Class I areas. Urban area annual mean and 98th percentile 24-hour average $PM_{2.5}$ levels for 1999 are presented above in Chapter 2. These levels are generally higher than those found in the IMPROVE database for rural Class I areas. In general, nitrates are responsible for a greater contribution to urban fine particle mass than in non-urban areas. In addition, some urban areas have higher concentrations of organic carbon and elemental carbon than rural areas due to a higher density of fuel combustion and diesel emissions.

5.2.4.1 Urban Visibility and $PM_{2.5}$ Monitoring Data

In the next draft of the Staff Paper, we intend to include information characterizing urban visibility for several cities around the country. Urban visibility data is available from the IMPROVE network for Washington, DC and South Lake Tahoe. Other cities with available visibility data include Denver, Phoenix, Seattle, and Tucson. In addition, as monitoring data become available from $PM_{2.5}$ speciation sites, we anticipate being able to calculate visibility for these sites in much the same way that is done for IMPROVE network sites.

5.2.4.2 ASOS Airport Visibility Monitoring Network

In 1992, the National Weather Service, Federal Aviation Administration, and Department of Defense began deployment of the Automated Surface Observing System (ASOS). ASOS is now the largest instrument-based visibility monitoring network in the U.S. (CD, 4-99). The ASOS visibility monitoring instrument is a forward scatter meter that has been found to correlate well with light extinction measurements from the Optec transmissometer (NWS, 1998). It is designed to provide consistent, real-time visibility and meteorological measurements to assist with air traffic control operations. More than 500 instruments have been commissioned and another 500 are planned for deployment in the coming years. ASOS visibility data is typically reported for aviation use in small increments up to a maximum of 10 miles visibility. While these truncated data are not useful for characterizing actual visibility levels, the raw, non-truncated data from the 1-minute light extinction and meteorological readings are now archived and available for analysis.

5.2.4.3 ASOS Data: Urban Visibility and Correlation to $PM_{2.5}$ Mass

To improve characterizations of current visibility conditions in non-class I areas, particularly in urban areas, EPA has obtained archived 1999 ASOS data for 63 cities across the country. Staff is in the process of analyzing the ASOS data to determine annual average, seasonal, monthly, and daily visibility conditions; best (10^{th} percentile) and worst (90^{th} percentile) day conditions; and diurnal and day of week conditions. Staff also plans to evaluate correlations between daily ASOS visibility data and 1999 24-hour $PM_{2.5}$ ambient monitoring data for a number of cities. Figure 5-2 is shown here as an illustrative example of such correlations. This information is expected to provide a better understanding of the average amount of light extinction per microgram of $PM_{2.5}$ in different parts of the country. Staff intends to include the results from these analyses in the next draft of this Staff Paper.

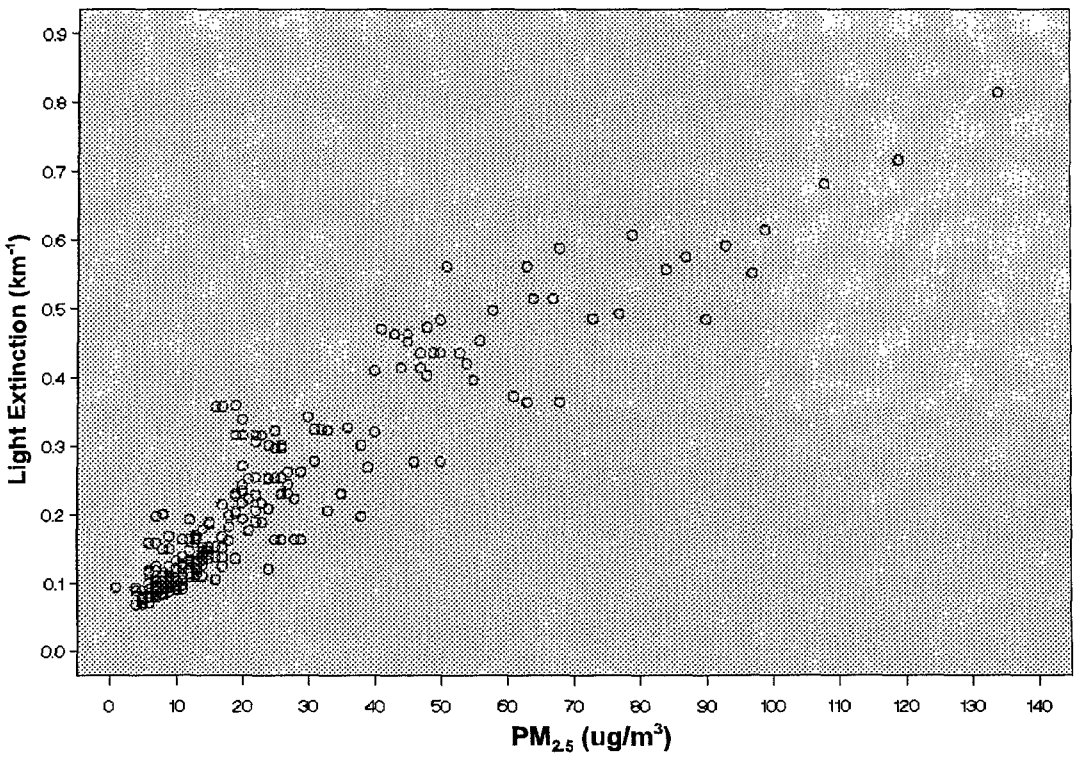

Figure 5-2. Correlation Between 1999 ASOS Airport Visibility Data (km^{-1}) and 24-Hour $PM_{2.5}$ Mass for Fresno, CA

5.2.5 Significance of Visibility to Public Welfare

Visibility is an air quality-related value having direct significance to people's enjoyment of daily activities in all parts of the country. Survey research on public awareness of visual air quality using direct questioning typically reveals that 80% or more of the respondents are aware of poor visual air quality (Cohen et al., 1986). The importance of visual air quality to public welfare across the country has been demonstrated by a number of studies designed to quantify the benefits (or willingness to pay) associated with potential improvements in visibility. More recently, the importance of visual air quality to the policymakers and the general public alike has also been demonstrated by a number of regional, state, and local efforts to address visibility impairment in urban and non-urban areas.

5.2.5.1 The Value of Improving Visual Air Quality

Individuals value good visibility for the well-being it provides them directly, both in the places where they live and work, and in the places where they enjoy recreational opportunities. Millions of Americans appreciate the scenic vistas in national parks and wilderness areas each year. Visitors consistently rate "clean, clear air" as one of the most important features desired in visiting these areas (Department of Interior, 1998). A 1998 survey of 590 representative households by researchers at Colorado State University found that 88% of the respondents believed that "preserving America's most significant places for future generations" is very important, and 87% of the respondents supported efforts to clean up air pollution that impacts national parks (Hass, 1998).

Economists have performed many studies in an attempt to quantify the economic benefits associated with improvements in current visibility conditions both in national parks and in urban areas. Economists distinguish between use values and non-use values. Use values are those aspects of environmental quality that directly affect an individual's welfare. These include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and hiking.

Non-use values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit. The component of non-use value that is related to the use of the resource by others in the future is referred to as the bequest value. This value is typically thought of as altruistic in nature. Another potential component of non-use value is the value that is related to preservation of the resource for its own sake, even if there is no human use of the resource. This component of non-use value is sometimes referred to as existence value or preservation value. Non-use values are not traded, directly or indirectly, in markets. For this reason, the measurement of non-use values has proved to be significantly more difficult than the measurement of use values. Non-use values may be related to the desire that a clean environment be available for the use of others now and in the future, or may be related to the desire to know that the resource is being preserved for its own sake, regardless of human use. Non-use values may be a more important component of value for recreational areas, particularly national parks and monuments.

It is well recognized in the U.S. and abroad that there is an important relationship between good air quality and economic benefits due to tourism. A 1998 study by the Department of Interior study found that travel-related expenditures by national park visitors alone average $14.5 billion annually (1996 dollars) and support 210,000 jobs (Peacock, 1998). A similar estimate of economic benefits resulting from visitation to national forests and other public lands could increase this estimate significantly.

McNeill and Roberge (2000) studied the impact of poor visibility episodes on tourism revenues in Greater Vancouver and the Lower Fraser Valley in British Columbia as part of the Georgia Basin Ecosystem Initiative of Environment Canada. Through this analysis a model was developed that predicts future tourist revenue losses that would result from a single extreme visibility episode. They found that such an episode would result in a $7.45 million loss in the Greater Vancouver area and $1.32 million loss in the Fraser Valley.

The results of several valuation studies addressing both urban and rural visibility are presented in the 1996 Criteria Document (EPA, 1996a, p. 8-83, Table 8-5; p. 8-85, Table 8-6) and in the 1996 Staff Paper (EPA, 1996b, p. VIII-3a, Table VIII-1; Chestnut et al., 1994). Past studies by Schulze (1983) and Chestnut and Rowe (1990b) have estimated the preservation values associated with improving the visibility in national parks in the Southwest to be in the range of approximately $2-6 billion annually (CD, 8-84). An analysis of the residential visibility benefits in the eastern U.S. due to reduced sulfur dioxide emissions under the acid rain program suggests an annual value of $2.3 billion (in 1994 dollars) in the year 2010 (Chestnut and Dennis, 1997). The authors suggest that these results could be as much as $1-2 billion more because the above estimate does not include any value placed on eastern air quality improvements by households in the western U.S.

Estimating benefits for visibility can be difficult because visibility is not directly or indirectly valued in markets. The studies cited above are based on a valuation method known as contingent valuation. Concerns have been identified about the reliability of value estimates from contingent valuation studies because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring willingness-to-pay for avoided health and welfare losses depends on the reliability and validity of the data collected.

However, there is an extensive scientific literature and body of practice on both the theory and technique of contingent valuation. EPA believes that well-designed and well-executed contingent valuation studies are useful for estimating the benefits of environmental effects such as improved visibility (EPA, 2000).

Society also values visibility because of the significant role it plays in transportation safety. Serious episodes of visibility impairment can increase the risk of unsafe air transportation, particularly in urban areas with high air traffic levels (EPA, 1982b). In some cases, extreme haze episodes have led to flight delays or the shutdown of major airports, resulting in economic impacts on air carriers, related businesses, and air travelers. For example, 24-hour $PM_{2.5}$ levels reached 68 $\mu g/m^3$ in St. Louis on May 15, 1998 during a haze episode attributed to wildfires in central America. This event resulted in a reduction in landing rates and significant flight delays at Lambert International Airport. In other cases, high $PM_{2.5}$ and haze levels, such as those experienced during the July 1999 air pollution episode in the northeastern U.S., have played a role in air transportation accidents and loss of life. (NTSB, 2000). During this episode, 24-hour levels of $PM_{2.5}$ ranged from 35-52 $\mu g/m^3$ in the New England states.

5.2.5.2 Visibility Goals and Programs

The value placed on protecting visual air quality is further demonstrated by the existence of a number of programs, goals, standards, and planning efforts that have been established in the U.S. and abroad to address visibility concerns in urban and non-urban areas. These regulatory and planning activities are of particular interest here to the extent that they are illustrative of the significant value that the public places on improving visibility, and because they have developed approaches and methods for evaluating public perceptions and judgments about the acceptability of varying degrees of visibility impairment that can be applied to develop additional information to help inform this review of the secondary PM NAAQS. Specific discussion is provided below on the statutory focus on visibility impairment in the U.S. Clean Air Act (CAA) and on the methods for evaluating public perceptions and judgments developed in conjunction with the establishment of a visibility standard in Denver.

Other examples of regulatory and planning activities in the U.S. include the establishment of visibility standards by the State of California (California Code of Regulations) and the Lake

Tahoe Regional Planning Agency (Molenar, 2000), and the initiative known as the Governor's Brown Cloud Summit in Phoenix, Arizona, for the future establishment of citizen-defined visibility goals using a citizen survey process similar to the Denver approach (Arizona Department of Environmental Quality, 2001).[2] International activities include the establishment of a visibility objective in the Australian state of Victoria (State Government of Victoria, 2000a and 2000b), the ongoing development of a visibility guideline in New Zealand (New Zealand National Institute of Water & Atmospheric Research, 2000a and 2000b; New Zealand Ministry of Environment, 2000), and field studies undertaken to characterize visibility and ambient aerosol loadings in southwestern British Columbia (Pryor, 1996), based on the methodology used by Ely et al. (1991) in setting the Denver visibility standard.

Sections 169A and 169B of the CAA. In addition to the recognition in sections 109 and 302(h) of the CAA that visibility impairment is a welfare effect that is to be protected by secondary NAAQS, additional protection of visibility impairment was outlined in sections 169A and 169B of the Act. Section 169A of the 1977 CAA Amendments established a national visibility goal to "remedy existing impairment and prevent future impairment" in 156 national parks and wilderness areas (Class I areas). The Amendments also called for EPA to issue regulations requiring States to develop long-term strategies to make "reasonable progress" toward the national goal. EPA issued initial regulations in 1980 focusing on visibility problems that could be linked to a single source or small group of sources. At this time, EPA deferred action on regional haze until monitoring, modeling, and source apportionment methods could be improved.

The 1990 CAA Amendments placed additional emphasis on regional haze issues through the addition of section 169B. In accordance with this section, EPA established the Grand Canyon Visibility Transport Commission (GCVTC) in 1991 to address adverse visibility impacts on 16 Class I national parks and wilderness areas on the Colorado Plateau. The GCVTC was comprised of the Governors of nine western states and leaders from a number of Tribal nations. The

[2]For illustrative purposes, Figures 27 to 34 in Appendix B show visual air quality in Phoenix under a range of visibility conditions. The images were generated using the WinHaze program, version 2.8.0, a state-of-the-art image modeling program developed by Air Resource Specialists, Inc.

GCVTC issued its recommendations to EPA in 1996, triggering a requirement in section 169B for EPA issuance of regional haze regulations.

EPA promulgated the final regional haze rule in 1999. The rule was developed with the benefit of many years of visibility research. Two key reports providing a technical basis for the rule were the 1991 NAPAP report and the 1993 National Academy of Sciences report on visibility in national parks and wilderness areas. The latter report concluded that "current scientific knowledge is adequate and control technologies are available for taking regulatory action to improve and protect visibility" (National Research Council, 1993).

Under the regional haze program, States are required to establish goals for improving visibility on the 20% most impaired days in each class I area, and for allowing no degradation on the 20% least impaired days. Each state must also adopt emission reduction strategies which, in combination with the strategies of contributing States, assure that class I area visibility improvement goals are met. The first State implementation plans are to be adopted in the 2003-2008 time period, with the first implementation period extending until 2018. Five multistate planning organizations are evaluating the sources of $PM_{2.5}$ contributing to Class I area visibility impairment to lay the technical foundation for developing strategies coordinated among many States in order to make reasonable progress in Class I areas across the country.

Denver Visibility Program and Standard-Setting Methodology. The State of Colorado adopted a visibility standard for the city of Denver in 1990.[3] Of particular interest here is the process by which the Denver visibility standard was developed, which relied on citizen judgments of acceptable and unacceptable levels of visual air quality (Ely et al., 1991).

Representatives from the Colorado Department of Public Health and Environment (CDPHE) conducted a series of meetings with 17 civic and community groups in which a total of 214 individuals were asked to rate slides having varying levels of visual air quality for a well-known vista in Denver. The CDPHE representatives asked the participants to base their judgments on three factors: 1) the standard was for an urban area, not a pristine national park area

[3] The Denver standard is violated when the four-hour average light extinction exceeds 76 Mm-1 (equivalent to approximately 32 miles visual range and 20 deciviews) during the hours between 8 a.m. and 4 p.m. Transmissometer readings taken when relative humidity is greater than 70% are excluded.

where the standards might be more strict; 2) standard violations should be at visual air quality levels considered to be unreasonable, objectionable, and unacceptable visually; and 3) judgments of standards violations should be based on visual air quality only, not on health effects.

The participants were shown slides in 3 stages. First, they were shown seven warm-up slides describing the range of conditions to be presented. Second, they rated 25 randomly-ordered slides based on a scale of 1 (poor) to 7 (excellent), with 5 duplicates included. Third, they were asked to judge whether the slide would violate what they would consider to be an appropriate urban visibility standard (i.e. whether the level of impairment was "acceptable" or "unacceptable").

The Denver visibility standard-setting process produced the following findings:

- Individuals' judgments of a slide's visual air quality and whether the slide violated a visibility standard are highly correlated (Pearson correlation coefficient greater than 80%) with the group average.

- When participants judged duplicate slides, group averages of the first and second ratings were highly correlated.

- Group averages of visual air quality ratings and "standard violations" were highly correlated. The strong relationship of standard violation judgments with the visual air quality ratings is cited as the best evidence available from this study for the validity of standard violation judgments (Ely et al., 1991).

The ratings for each slide were sorted by increasing order of light extinction, and the percentage of participants that judged each slide to violate the "standard" was calculated. The Denver visibility standard was then established based on a 50% acceptability criterion. Under this approach, the standard was identified as the light extinction level that divides the slides into two groups: those found to be acceptable and those found to be unacceptable by a majority of study participants. For illustrative purposes, Figures 19 to 26 in Appendix B show visual air quality in Denver under a range of visibility conditions (generally corresponding to 10^{th}, 20^{th}, 30^{th}, 40^{th}, 50^{th}, 60^{th} 80^{th}, and 90^{th} percentile values). These images were generated using the WinHaze program, version 2.8.0, a state-of-the-art image modeling program developed by Air Resource Specialists, Inc.

5.2.6 Evaluating Public Perceptions of Visibility Impairment

New tools and methods are now available to communicate and evaluate public perceptions of varying visual effects associated with alternative levels of visibility impairment relative to varying pollution levels and environmental conditions. As described above in Section 5.2.5.2, these tools and methods have been used by others as a basis for developing goals and standards for visibility. Building upon this work, EPA has initiated a project to evaluate public perceptions of visibility impairment in urban areas, and intends to consider using the information developed in this project to help inform the review of the secondary PM NAAQS. In particular, new techniques for photographic representation of visibility impairment are discussed below, followed by a discussion of the survey approach used in the pilot phase of this project and the plans for the continuation of this project.

Staff welcomes CASAC and public input on the information presented below, including the photographic techniques and survey methods planned for use in this project, and the appropriateness of using the results from this project to help inform our review of the secondary PM NAAQS.

5.2.6.1 Photographic Representations of Visibility Impairment

In the past, the principal method for recording and describing visual air quality has been through 35 millimeter photographs. Under the IMPROVE program, EPA and its optical monitoring contractor Air Resource Specialists, Inc. (ARS) have developed an extensive archive of visual air quality photos for national parks and wilderness areas. In comparison, we have only a limited archive of photos of urban areas.

The draft CD discusses some of the methods that are now available to represent different levels of visual air quality (CD, p. 4-107). In 1994, Molenar described a sophisticated visual air quality simulation technique in Atmospheric Environment (Molenar, 1994). This technique, a combination of modeling systems under development for the past 20 years, was developed by ARS.

The technique relies on first obtaining an original base image slide of the scene of interest. The slide should be of a cloudless sky under the cleanest air quality conditions possible. The light extinction represented by the scene should be derived from aerosol and optical data associated

with the day the image was taken, or it should be estimated from contrast measurements of features in the image. The image is then digitized to assign an optical density to each pixel. At this point, the radiance level for each pixel is estimated. Using a detailed topographic map, technicians identify the specific location from which the photo was taken, and they determine the distances to various landmarks and objects in the scene. With this information, a specific distance and elevation is assigned to each pixel.

Using the digital imaging information above, the system then computes the physical and optical properties of an assumed aerosol mix. These properties are input into a radiative transfer model in order to simulate the optical properties of varying pollutant concentrations on the scene. ARS now provides WinHaze, version 2.8.0, an image modeling program for personal computers that employs simplified algorithms based on the sophisticated modeling technique developed by Molenar.

An alternative technique would be to obtain actual photographs of the site of interest at different ambient pollution levels. However, long-term photo archives of this type exist for only a few cities. In addition, studies have shown that observers will perceive an image with a cloud-filled sky as having a higher degree of visibility impairment than one without clouds, even though the PM concentration on both days is the same. The simulation technique has the advantage that it can be done for any location as long as one has a very clear base photo. In addition, the lack of clouds and consistent sun angle in all images in effect standardizes the perception of the images and enables researchers to avoid potentially biased responses due to these factors.

5.2.6.2 Pilot Project: Assessing Public Opinions on Air Pollution-Related Visibility Impairment

The pilot project described here uses the latest techniques for photographic representation of visibility impairment and survey techniques applied by others as a basis for setting visibility goals and standards. Staff developed this project to provide information that may be useful in the EPA's review of the secondary PM NAAQS. The project is premised on the view that public perceptions of and judgments about the acceptability of visibility impairment in urban areas are relevant factors in assessing what constitutes an adverse level of visibility impairment in the context of this NAAQS review.

With this in mind, staff considered various approaches for obtaining public input on visibility impairment. Potential options included a mail survey, a web-based computer survey, a computer-based survey in a public location, and face-to-face meetings with survey participants. As discussed below, one important issue that staff considered in selecting a preferred option involved how to develop images that graphically represent subtle differences in pollutant concentrations and air quality, and selecting the appropriate media for communicating these images to public citizens. Another issue was how to ensure consistency in the way in which participants in any such survey would receive and process this information, recognizing that the method used to conduct the survey (e.g., mail delivery, presentations to small groups) could affect this consistency since the methods differ in the extent of control that the researchers have of the survey process.

Developing Images. The options for presenting images include web-based digital images viewed on computer monitors, print photos, video or DVD, and 35 millimeter slides. Thirty-five millimeter slides generally provide the highest resolution, and the researcher can have a high level of control in how they are presented. As discussed above, this approach was used by Colorado Department of Public Health and Environment staff in its research leading to development of the Denver visibility standard. Large format print photos also have high resolution, but are more costly than slides. The best quality computer monitors can also provide high resolution, but resolution varies greatly from monitor to monitor if the images were provided on the internet. Creating multiple copies of print photos to accompany a mail survey would be quite expensive,

and there would be little control in how the photos would be presented. Taking all of this into account, staff decided to use high resolution 35 mm slides presented to a small group of people at a time.

Having made this decision on image media, staff decided to pursue a pilot project similar to the Denver study that used the ARS visual air quality modeling technique to communicate different levels of visibility impairment to members of the general public. EPA contracted with ARS to develop a series of 27 images of a scene in Washington, DC, consistent with the approach described above. ARS developed this slide series for a vista of Washington, DC as viewed from across the Potomac River near Arlington Cemetery. The vista includes the Mall in downtown Washington, DC and several well-known landmarks, including the Lincoln Memorial, Washington Monument, Capitol Building, Union Station, and Library of Congress. The sight path to the farthest landmark in the scene (the Anacostia neighborhood) is fairly short – approximately 8 km. The base image was taken on a clear day with no cloud cover.

The slides illustrate visual air quality associated with $PM_{2.5}$ concentrations across a broad range of possible conditions, ranging from 2.3 $\mu g/m^3$ to 65 $\mu g/m^3$. Figures 6 and 10 in Appendix B show Washington, DC at 15 $\mu g/m^3$ and 65 $\mu g/m^3$ levels, respectively. The same pollutant mix was used to make each slide so that changes in visual air quality from slide to slide could be attributed solely to changes in PM mass concentrations. For each image, the percent of total $PM_{2.5}$ mass assigned to each component was chosen based on annual average values derived from data collected at the Washington, DC IMPROVE monitoring site from 1988 to 1999. For each $PM_{2.5}$ level, the assumed pollutant mix was as follows: sulfate = 50%; nitrate = 10%; organic carbon = 25%; elemental carbon = 10%; fine soil = 5%.

Coarse-fraction particles also cause light scattering, but are less efficient per unit mass. Based on the relationship of PM_{10} and $PM_{2.5}$ values from Washington, DC IMPROVE data (1988-99), a standard mass value was assigned to PM_{10} for each image equal to 30 % of the $PM_{2.5}$ mass. A standard value of 10 Mm-1 was assumed for Rayleigh scattering. Light absorption by gases is commonly attributed to NO_2, which gives a brownish cast to the sky color, particularly in urban areas. Based on a review of recent AIRS data for Washington, DC, an annual average value of 16 ppb was assumed for NO_2 and taken into account in the image modeling process. Finally, the

images were generated using an assumed annual average relative humidity of 68% (corresponding to an f(RH) factor of 2.98 for calculating light extinction due to sulfates and nitrates). This annual average relative humidity value was derived from National Weather Service data from nearby airports.

Appendix B includes the specific data and the photographic images used in the pilot survey. In particular, Tables 1 and 2 in Appendix B provide the pollutant concentrations and the calculated visibility parameters (i.e., light extinction, visual range, and deciviews), respectively, used to create each slide. Figures 3 through 10 in Appendix B display images of Washington, DC representing 24-hour $PM_{2.5}$ levels of 2.5, 5, 10,15, 20, 30, 40, and 65 $\mu g/m^3$, respectively. Series of images are also provided in Appendix B for Chicago, Illinois (Figures 11-16), Denver, Colorado (Figures 19-26), and Phoenix, Arizona (Figures 27-34).

Focus Group Process and Pilot Survey. EPA contracted with Abt Associates to coordinate the implementation of a pilot focus group session, held on November 16, 2000 in Bethesda, Maryland. The session was designed based on the approach used for the Denver study (see Section 5.2.2.2 above and Ely et al., 1991).[4] This same approach has been successfully implemented by other researchers as well (Pryor, 1996; Hill et al., 2000). The purpose of the pilot focus group session was to evaluate the initial survey process and survey questions so as to refine the approach for future sessions to be held in different cities around the country. Abt Associates summarized the conduct and results from the pilot focus group session in a January 2001 report (Abt Associates, 2001). This report is available for review.

More specifically, six female and three male participants from Maryland, Virginia, and the District of Columbia were invited to participate in the session. Demographically, the group represented a balanced range of ages, races, education levels, and income levels. The session was held in a large meeting room with a one-way mirror for observation by EPA and Abt representatives. Two representatives from Abt Associates facilitated the session. The 35 mm

[4] Methods for the Denver study were based on previous research conducted by the National Park Service (Malm et al., 1981) and National Center for Atmospheric Research (Stewart et al., 1983). The results from these studies have shown that judgments of visual air quality by private citizens are valid and reliable. They also have shown that judgments made from one group to another are highly correlated, and that judgments made from slides are highly correlated to those made in the field (Ely et al., 1991).

slides were displayed on an eight-foot matte screen using a Kodak AMT Ektagraphic projector with a high quality projection lens (f2.8). The participants were located approximately 9 to 13 feet from the projection screen.

The session involved viewing slides in three steps as discussed in the overview of the Denver study. In designing the session, representatives from EPA and Abt Associates decided that to address time constraints and the subtlety of changes between some of the slides with higher $PM_{2.5}$ concentrations, a subset of the 25 slides should be shown. Accordingly, a set of 20 of the 25 original slides were selected for the pilot session. Five duplicates were selected at random and added to the set of 20 originals, resulting in a total set of 25 slides.

The participants were first shown a series of four "warm-up" slides representing the full range of visual air quality conditions they were about to view. Next, the participants were shown the 25 slides in random order and asked to rate the visual air quality of each slide on a seven-point scale, ranging from "Very Poor" to "Very Good." A cumulative score was calculated for each slide by assigning 1 (very poor) to 7 (very good) points to each participant's response, with 63 being the highest cumulative score a slide could receive from the group. Based on the results, it appears that the participants were able to perceive subtle differences between slides in a consistent manner. The cumulative scores for each slide are shown in Figure 17 in Appendix B.

In the final step of the rating process, the participants viewed the slides in a random order again, and were asked to rate the slide as "acceptable" or "unacceptable." They were asked to consider only the visual air quality of the scene, not any assumed public health consequences, nor the potential costs of improving conditions to an "acceptable" level. The results showed three distinct "zones" resulting from the rating process:

- "Acceptable" zone: the set of slides found to be "acceptable" by most participants. (In this case, the acceptable zone generally included slides for 15 $\mu g/m^3$ and less.)

- "Unacceptable" zone: the set of slides found to be "unacceptable" by most participants. (In this case, the unacceptable zone generally included slides for 40 $\mu g/m^3$ and above.)

- "Intermediate" zone: the remaining set of slides, for which there were varying degrees of "acceptable" and "unacceptable" ratings.

Figure 18 in Appendix B illustrates the number of respondents who rated each slide as acceptable or unacceptable. This basic pattern of responses is similar to that found in the Denver study. Staff expects that the results from future meetings to obtain citizen input will also show three basic rating "zones." One objective of a broader survey of citizens will be to see if the $PM_{2.5}$ levels shaping these zones are relatively consistent or highly variable from one region of the country to another.

After the slide rating portion of the session, EPA staff joined the group for a discussion to evaluate the session design. In this part of the session, staff reviewed the survey questions with the participants to determine whether some questions were difficult to understand and needed clarification. We also asked the participants to comment on whether they took health effects or weather effects into account in the rating process. Regarding health effects, staff purposefully designed the survey questions to emphasize that the visual air quality (VAQ) ratings should be based only on the participant's judgment of the visibility level, and should not involve any assumptions about negative health effects that might be experienced from such a VAQ level. The respondents agreed that the survey should not take health effects into account since this could lead to biased responses. Regarding weather effects, some participants stated that some of the hazier images looked like there was a heavy fog present. It was recommended that in future sessions, the facilitator should emphasize that the weather condition in each slide is the same (e.g. a cloudless day), with no fog or precipitation in the air. The summary report for the pilot session includes discussion of a number of other questions asked during the session and potential design improvements (Abt Associates, 2001).

Planned Focus Group Survey. During 2001-2002, staff is planning to conduct additional survey sessions to obtain citizen input on visual air quality in New York City; Asheville, NC; Chicago; Seattle; San Francisco; and at least one other western city to be determined. EPA has contracted with ARS for the development of a high quality slide series for each of these cities. EPA intends to contract with a consulting firm to coordinate the sessions, as was done for the pilot session. The purpose of these additional citizen input sessions will be to evaluate the consistency of citizen responses from one region of the country to another.

5.3 EFFECTS ON MATERIALS

The effects of the deposition of atmospheric pollution, including ambient PM, on materials are related to both physical damage and aesthetic qualities. The deposition of PM (especially sulfates and nitrates) can physically affect materials, adding to the effects of natural weathering processes, by potentially promoting or accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Particles contribute to these physical effects because of their electrolytic, hygroscopic and acidic properties, and their ability to sorb corrosive gases (principally SO_2). As noted in the last review, only chemically active fine-mode or hygroscopic coarse-mode particles contribute to these physical effects (EPA 1996b, p. VIII-16).

In addition, the deposition of ambient PM can reduce the aesthetic appeal of buildings and culturally important articles through soiling. Particles consisting primarily of carbonaceous compounds cause soiling of commonly used building materials and culturally important items such as statues and works of art (CD, p. 4-114). Soiling is the deposition of particles on surfaces by impingement, and the accumulation of particles on the surface of an exposed material results in degradation of its appearance. Soiling can be remedied by cleaning or washing, and depending on the soiled material, repainting (EPA, 1996b, p. VIII-19).

Building upon the information presented in the last Staff Paper (EPA, 1996b), and including the limited new information presented in Chapter 4 of the draft CD, the following sections summarize the physical damage and aesthetic soiling effects of PM on materials including metals, paint finishes, and stone and concrete.

5.3.1 Materials Damage Effects

Physical damage such as corrosion, degradation, and deterioration occurs in metals, paint finishes, and building materials such as stone and concrete, respectively. Metals are affected by natural weathering processes even in the absence of atmospheric pollutants. Atmospheric pollutants, most notably SO_2 and particulate sulfates, can have an additive effect, by promoting and accelerating the corrosion of metals. The rate of metal corrosion depends on a number of factors, including the deposition rate and nature of the pollutants; the influence of the protective

corrosion film that forms on metals, slowing corrosion; the amount of moisture present; variability in electrochemical reactions; the presence and concentration of other surface electrolytes; and the orientation of the metal surface. Historically, studies have shown that the rate of metal corrosion decreases in the absence of moisture, since surface moisture facilitates the deposition of pollutants and promotes corrosive electrochemical reactions on metals.

The draft CD (p. 4-117, Table 4-8) summarizes the results of a number of studies investigating the roles of particles (e.g., particulate sulfates) and SO_2 on the corrosion of metals. The draft CD concludes that the role of particles in the corrosion of metals is not clear (CD, p. 4-116). While several studies suggest that particles can promote the corrosion of metals, others have not demonstrated a correlation between particle exposure and metal corrosion. Although the corrosive effects of SO_2 exposure in particular have received much study, there remains insufficient evidence to relate corrosive effects to specific particulate sulfate levels or to establish a quantitative relationship between ambient particulate sulfate and corrosion.

Similar to metals, paints also undergo natural weathering processes, mainly from exposure to environmental factors such as sunlight, moisture, fungi, and varying temperatures. Beyond these natural processes, atmospheric pollutants can affect the durability of paint finishes by promoting discoloration, chalking, loss of gloss, erosion, blistering, and peeling. Historical evidence indicates that particles can damage painted surfaces by serving as carriers of more corrosive pollutants, most notably SO_2, allowing the pollutants to reach the underlying surface, or by serving as concentration sites for other pollutants. A number of studies available in the last review showed some correlation between PM exposure and damage to automobile finishes. In particular, Wolff et al. (1990) concluded that damage to automobile finishes resulted from calcium sulfate forming on painted surfaces by the reaction of calcium from dust particles and sulfuric acid contained in rain or dew. In addition, paint films permeable to water are also susceptible to penetration by acid forming aerosols (EPA 1996b, p. VIII-18). The erosion rate of oil-based house paint has been reported to be enhanced by exposure to SO_2 and humidity; several studies have suggested that the effect of SO_2 is caused by its reaction with extender pigments such as calcium carbonate and zinc oxide, although Miller et al. (1992) suggests that calcium carbonate acts to protect paint substrates (CD, p. 4-119).

With respect to damage to building stone, numerous studies discussed in the draft CD (p. 4-120, Table 4-9) suggest that air pollutants, including sulfur-containing pollutants and atmospheric particles including gypsum, can enhance natural weathering processes. Exposure-related damage to building stone results from the formation of salts in the stone that are subsequently washed away by rain, leaving the surface more susceptible to the effects of air pollutants. Dry deposition of sulfur-containing pollutants and carbonaceous particles promotes the formation of gypsum on the stone's surfaces. Gypsum is a black crusty material that occupies a larger volume than the original stone, causing the stone's surface to become cracked and pitted, leaving rough surfaces that serve as sites for the deposition of airborne particles (CD, page 4-124).

The rate of deterioration of building stone is determined by the pollutant mix and concentration, the stone's permeability and moisture content, and the pollutant deposition velocity. Dry deposition of SO_2 between rain events has been reported to be a major causative factor in pollutant-related erosion of calcareous stones (e.g., limestone, marble, and carbonated cement). While it is clear from the available information that gaseous air pollutants, in particular SO_2, will promote the decay of some types of stones under specific conditions, carboneous particles (non-carbonate carbon) and particles containing metal oxides may help to promote the decay process (CD, p. 4-125).

5.3.2 Soiling Effects

Soiling affects the aesthetic appeal of painted surfaces, including culturally important articles, and stone surfaces. In addition to natural factors, exposure to PM may give painted surfaces a dirty appearance, although few studies are available that evaluate the soiling effects of particles (CD, p. 4-127). Early studies demonstrated an association between particle exposure and increased frequency of cleaning painted surfaces. More recently, Haynie and Lemmons (1990) conducted a study to determine how various environmental factors contribute to the rate of soiling on white painted surfaces. They reported that coarse-mode particles initially contribute more to soiling of horizontal and vertical surfaces than do fine-mode particles, but are more easily removed by rain, leaving stains on the painted surface. The authors concluded that the

accumulation of fine-mode particles, rather than coarse-mode particles, more likely promotes the need for cleaning of the painted surfaces (EPA 1996b, p. VIII-21-22). Creighton et al. (1990) reported that horizontal surfaces soiled faster than vertical surfaces and that large particles were primarily responsible for the soiling of horizontal surfaces not exposed to rainfall. Additionally, a study was conducted to determine the potential soiling of artwork in five Southern California museums (Ligocki, et al., 1993). Findings were that a significant fraction of fine elemental carbon and soil dust particles in the ambient air had penetrated to the indoor environment and may constitute a soiling hazard to displayed artwork (EPA 1996b, p. VIII-22).

As for stone structures, the presence of gypsum is related to soiling of the stone surface by providing sites for particles of dirt to concentrate. Lorusso et al. (1997) attributed the need for frequent cleaning and restoration of historic monuments in Rome to exposure to total suspended particles (TSP). Further, Davidson et al. (2000) evaluated the effects of air pollution exposure on a limestone structure on the University of Pittsburgh campus using estimated average TSP levels in the 1930s and 1940s and actual values for the years 1957 to 1997. Monitored levels of SO_2 were available for the years 1980 to 1998. Based on the available data on pollutant levels and photographs, it was thought that soiling began while the structure was under construction. With decreasing levels of pollution, the soiled areas have been slowly washed away, the process taking several decades, leaving a white, eroded surface (CD, pages 4-126 to 4-127).

5.3.4 Summary

Damage to building materials results from natural weathering processes that are enhanced by exposure to airborne pollution, most notably sulfur-containing pollutants. While ambient PM has been associated with contributing to pollution-related damage to materials, the draft CD concludes that insufficient data exist to relate such effects to specific particle pollution levels, particle size, or chemical composition (CD, p. 4-163). In addition to contributing to physical damage, particle pollution can cause significant detrimental effects by soiling painted surfaces and other building materials. Available data indicate that particle-related soiling can result in increased cleaning frequency and repainting, and may reduce the useful life of the soiled materials.

However, again the draft CD concludes that insufficient data are available to relate soiling effects to specific particle pollutant levels, particle size, or chemical composition (CD, p.4-163).

5.4 EFFECTS ON VEGETATION AND ECOSYSTEMS

Environmental impacts of ambient PM are considered here in relation to effects on vegetation and other components of the environment, such as soils, water, and wildlife, that make up ecosystems. Observed effects can result from the physical and chemical properties of PM and may be caused directly by particle deposition onto the affected vegetation or indirectly through deposition to soils or water. However, the draft CD notes that particle deposition to vegetation and ecosystems is not well understood at this time (CD, p. 4-2). Available evidence does suggest that all modes of deposition must be considered in determining potential impacts to vegetation and ecosystems including: 1) wet deposition in which particles are deposited in rain and snow; 2) occult deposition in which particles are deposited in fog, cloud-water and mists; and 3) dry deposition in which particles are deposited onto surfaces (CD, p. 4-3). Wet deposition is generally more effective for removing fine-mode PM from the atmosphere, whereas dry deposition is more effective for coarse-mode particles.

Based on information contained and referenced in Chapter 4 of the draft CD, the effects of ambient PM alone and in combination with other pollutants are summarized below, focusing first on direct effects on vegetation, then more broadly and importantly on direct and indirect effects on ecosystems.

5.4.1 Direct Effects on Vegetation

Particulate matter that deposits directly from the atmosphere onto above-ground plant surfaces may (1) reside on the leaf, twig, or bark surface for an extended period; (2) be taken up through the leaf surface; or 3) be removed from the plant via resuspension to the atmosphere, washing off by rainfall, or litter-fall with subsequent transfer to the soil (CD, p.4-6). The following discussion focuses on those particles that are intercepted by and remain on the leaves. Most information currently available on plant effects focuses on nitrate particle deposition, in particular, and more generally on acidic deposition, primarily from nitrogen- and sulfur-

containing particles and gaseous pollutants. Depending on the amount and composition of the deposited PM, effects can be either physical, chemical, or both.

Physical effects of PM occur mainly in areas where deposition rates for particles in the coarse mode are high, in some cases leading to crust formation on plant leaves, such as near roadways, agricultural areas and industrial sites. Physical effects that have been observed in vegetation in such areas include reduced photosynthesis and subsequent reductions in carbohydrate formation, root and plant growth; blockage of the stomata preventing adequate gas exchange; changes in leaf temperature (e.g., heat stress); destruction of leaf tissue (e.g., chlorosis, necrosis, and/or abscission); and premature leaf-fall. (CD, pp. 4-7 to 4-8).

In most areas, however, where deposition rates are not high enough for significant physical effects from PM to occur, the chemical composition of PM becomes the key phytotoxic factor leading to plant injury. Often, it is the chemical composition or class of PM in the fine mode that produces phytotoxic effects when deposited onto plant surfaces, as discussed below first for nitrates and other acidic particles, and then for trace metals and organics. However, studies of the direct effects of chemical additions to foliage through particle deposition have found little or no effects of PM on foliar processes unless exposure levels were significantly higher than typically would be experienced in the ambient environment. Further, only a few studies have been completed on the direct effects of fine-mode particles on vegetation, and the conclusion that was reached in the 1982 PM Criteria Document (EPA, 1982), that sufficient data were not available for adequate quantification of dose-response functions, continues to be true today (CD, pp. 4-6 to 4-9).

Acidic Deposition. Nitrogen has long been recognized as the nutrient most important for plant growth. For instance, approximately 75% of the nitrogen in a plant leaf is used during the process of photosynthesis, and to a large extent, it governs the utilization of phosphorus, potassium, and other nutrients. Particle deposition of nitrate, together with other nitrogen-containing gaseous and precipitation-derived sources, represent a substantial fraction of total nitrogen reaching vegetation. However, much of this nitrogen is contributed by gaseous nitric acid vapor, and a considerable amount of the particulate nitrate is taken up indirectly through the soil (CD, p. 4-9). Though plants usually absorb nitrogen (as NH_4^+ or NO_3^-) through their roots,

it is known that foliar uptake of nitrate can occur. However, the mechanism of foliar uptake is not well established, plants vary in their ability to absorb ammonium and nitrate, and it is not currently possible to distinguish sources of chemicals deposited as gases or particles using foliar extraction. Since it has proven difficult to quantify the percentage of nitrogen uptake by leaves that is contributed by ambient particles, direct foliar effects of nitrogen-containing particles have not been documented. (CD, pp. 4-10 to 4-11; 4-41 to 4-42).

Similar to nitrogen, sulfur is an essential plant nutrient that can deposit on vegetation in the form of sulfate particles, or be taken up by plants in gaseous form. Greater than 90% of anthropogenic sulfur emissions are as sulfur dioxide (SO_2), with most of the remaining emissions in the form of sulfate. However, sulfur dioxide is rapidly transformed in the atmosphere to sulfate, which is approximately 30-fold less phytotoxic than SO_2. Low dosages of sulfur can serve as a fertilizer, particularly for plants growing in sulfur-deficient soils. There are only a few field demonstrations of foliar sulfate uptake, however, and the relative importance of foliar leachate and prior dry-deposited sulfate particles remains difficult to quantify. Though current levels of sulfate deposition reportedly exceed the capacity of most vegetative canopies to immobilize the sulfur, sulfate additions in excess of needs do not typically lead to plant injury. Additional studies are needed, however, on the effects of sulfate particles on physiological characteristics of plants following chronic exposures (CD, pp. 4-11 to 4-12).

Though dry deposition of nitrate and sulfate particles does not appear to induce foliar injury at current ambient exposures, when found in acidic precipitation, they do have the potential to cause direct foliar injury. This is especially true when the acidic precipitation is in the form of fog and clouds, which may contain solute concentrations up to 10 times those found in rain. In experiments on seedling and sapling trees, both coniferous and deciduous species showed significant effects on leaf surface structures after exposure to simulated acid rain or acid mist at pH 3.5, while some species have shown subtle effects at pH 4 and above. Epicuticular waxes, which function to prevent water loss from plant leaves, can be destroyed by acid rain in a few weeks which suggests links between acidic precipitation and aging. Due to their longevity and evergreen foliage, the function of epicuticular wax is more crucial in conifers. For example, red spruce seedlings, which have been extensively studied, appear to be more sensitive to acid

precipitation (mist and fog) when compared with other species (CD, pp. 4-13 to 4-14). In addition to accelerated weathering of leaf cuticular surfaces, other direct responses of forest trees to acidic precipitation include increased permeability of leaf surfaces to toxic materials, water, and disease agents; increased leaching of nutrients from foliage; and altered reproductive processes (CD, p. 4-29). All of these effects serve to weaken trees so that they are more susceptible to other stresses (e.g., extreme weather, pests, pathogens).

Trace elements. Of the 90 elements that make up the inorganic fraction of the soil, 80 exist in concentrations of less than 0.1% and are known as "trace elements". Trace elements with a density greater than 6 g/cm^3 are referred to as "heavy metals". Although some trace metals are essential for vegetative growth or animal health, in large quantities, they are all toxic. Most trace metals found in the atmosphere are produced by industrial combustion processes and exist predominantly as metal chloride particles, which tend to be volatile, or as metal oxides, which tend to be nonvolatile and in the vapor phase. Heavy metals introduced into the atmosphere from human activities include antimony, cadmium, chromium, copper, lead, molybdenum, mercury, nickel, silver, tin, vanadium, and zinc (CD, p. 4-15).

Investigations of trace elements present along roadsides and in industrial and urban environments have indicated that impressive burdens of particulate heavy metal can accumulate on vegetative surfaces. Once on the surface, these metals can potentially impact either the metabolism of above-ground plant tissues or the activity of populations of organisms resident on and in the leaf surface (e.g., bacteria, fungi and arthropods). In the first scenario, a trace metal must be brought into solution before it can enter into the leaves or bark of vascular plants. Since the solubility of most trace metals is low, foliar uptake and direct heavy metal toxicity is limited. In those instances when trace metals are absorbed, they are frequently bound in leaf tissue and are lost when the leaf later drops off. Only a few metals have been documented to cause direct phytotoxicity in field conditions, with copper, zinc and nickel toxicities observed most frequently. It is unlikely, therefore, that deposition of trace metals to vegetative surfaces at ambient levels is causing wide spread acute plant toxicity. In the second scenario, little experimental data exists on the effects of trace metals on leaf surface organisms, though trace metal toxicity of lichens has been demonstrated in a few cases (CD, pp. 4-16 to 4-17).

On the other hand, the effects of chronic low-level metal deposition on perennial plant species may be more significant than the acute effects referred to above. When trees are exposed to sub-lethal concentrations of heavy metals, levels of intracellular metal-binding peptides, phytochelatins, increase. In studies designed to test the relationship between heavy metals and the decline of forest tree species in certain areas in the U.S., the data showed a systematic and significant increase in phytochelatin concentrations associated with the extent of tree injury. Though there has been no direct evidence of a physiological association between tree injury and exposure to metals, metals have been implicated because their deposition pattern has been correlated with the decline of certain tree species. (CD, pp. 4-16 to 4-17).

Organics. Many different chemical compounds can fall under the generic classification of "organics". These compounds may also be referred to as toxic substances, pesticides, hazardous air pollutants (HAPs), air toxics, semivolatile organic compounds (SOCs), and persistent organic pollutants (POPs). While these substances are not criteria pollutants, they are discussed here because many of these compounds partition between gas and particle phases and are removed from the atmosphere by both wet and dry deposition.. As particles they can become airborne, be distributed over wide areas, and impact remote ecosystems. Some notable organics include such compounds as DDT, polychlorinated biphenyls (PCBs), and polynuclear aromatic hydrocarbons (PAHs). These substances may enter plants via the roots, be deposited as particles onto the waxy cuticle of leaves or be taken up through the stomata. Which pathway is followed is a function of the chemical and physical properties of the pollutant, environmental conditions, and the plant species. However, the direct uptake of organic contaminants through the cuticle or in the vapor phase through the stomates are poorly characterized for most trace organics. Additionally, the toxicity of organic contaminants to plants and soil microorganisms is not well studied (CD, pp. 4-18 to 4-19).

5.4.2 Ecosystem Effects

As discussed in the draft CD, human existence on this planet depends on the life-support services ecosystems provide. Both ecosystem structure and function play essential roles in providing societal benefits, including products with market value (e.g., fish, minerals, forest

products, biomass fuels, natural fibers, pharmaceuticals) as well as the use and appreciation of natural areas for recreation, aesthetic enjoyment, and study. In addition, ecosystem functions play a major role in maintaining necessary atmospheric, climatic, and radiative balances within our environment (e.g., absorbing pollution, cycling nutrients, degrading wastes) (CD, p. 4-156). The draft CD provides a detailed discussion of the nature of ecosystems, the services they provide, and their response to stress (CD, pp. 4-20 to 4-25).

Ecosystem-level responses occur when the effects of particulate deposition on the biological and physical components of ecosystems become sufficiently widespread as to impact essential processes such as cycling of nutrients and materials. Such responses can be a result of physical effects caused by high levels of PM dust being deposited directly onto vegetative surfaces over a large portion of a plant community, or more importantly, from the chemical effects resulting from the chemical constituents of PM deposited directly onto vegetative surfaces or indirectly through deposition into soil and water environments.

Plant community structure is determined by sampling the various strata within the community (e.g., herbs, seedlings, saplings, trees). Long-term changes in the structure and composition of the strata within plant communities exposed to chronic dust accumulation have been observed, demonstrating that the physical effects of dust accumulation favors the growth of some species and limits others. Specifically, at an experimental site near limestone quarries and processing plants in southwestern Virginia, where dust accumulation occurred for at least 30 years, red maple was more abundant in all strata when compared with the control site where it was present only as a seedling. The growth of tulip poplar, dogwood, hop-hornbeam, black haw and red bud appeared to be favored by the dust, while the growth of conifers and other acid tolerant species such as rhododendron, was limited. It can be assumed that changes in soil alkalinity also occurred at the site due to the heavy deposition of limestone dust, but in the absence of soil analyses, no conclusion was reached as to the role that chemical changes to the soils may have played in these plant community changes. This site exemplifies how the direct physical effects of PM can impact ecosystems (CD, pp. 4-27 to 4-29).

Aside from its physical effects, the impact of PM on ecosystems is determined chiefly by its chemical constituents and their ability to affect the nutrient status of the ecosystem, either by

direct foliar uptake or by directly or indirectly changing soil chemistry, populations of bacteria involved in nutrient cycling, and/or populations of fungi involved in plant nutrient uptake (CD, p. 4-34).

Acidic Deposition. As discussed above, several of the chemical components of PM (e.g., nitrogen, sulfur, calcium) are essential plant nutrients. Additions of any of these nutrients, most importantly particulate nitrogen (nitrates), can affect plant succession patterns and biodiversity. Nitrogen has long been recognized as the nutrient most important for plant growth. In soils low in nitrogen, atmospherically deposited nitrogen can act as a fertilizer. However, not all plants are capable of utilizing extra nitrogen. Inputs of nitrogen to natural ecosystems that alleviate deficiencies and increase growth of some plants can impact competitive relationships and alter species composition and diversity. Plants growing in low resource environments (e.g., infertile soil, shaded understory, deserts, tundra) have been observed to have certain similar characteristics: 1) a slow growth rate, 2) low photosynthetic rate, and 3) low capacity for nutrient uptake (e.g., they tend to respond less than other plant species even when provided with an optimal supply and balance of resources). Since not all plants are equally capable of utilizing extra nitrogen, as nitrogen becomes more readily available, some plants will gain a competitive advantage and will replace those adapted to living in lower nitrogen environments (CD, pp. 4-45 to 4-46). For example, Fenn et al. (1998) report that long-term nitrogen fertilization studies in both New England and Europe suggest that some forests receiving chronic inputs of nitrogen may decline in productivity and experience greater mortality. Long-term fertilization experiments at Mount Ascutney, Vermont, suggest that declining coniferous forest stands with slow nitrogen cycling may be replaced by deciduous fast-growing forest species that cycle nitrogen rapidly (Fenn et al., 1998; CD, p. 4-47).

In some cases, additions of nitrogen above soil background levels can exceed the capacity of plants and soil microorganisms to utilize and retain it, resulting in a condition known as "nitrogen saturation." Specific ecosystem processes affected by nitrogen saturation include: 1) increased plant uptake and allocation, (i.e., a permanent increase in foliar nitrogen and reduced foliar phosphorus and lignin due to the lower availability of carbon, phosphorus, and water); 2) increased litter production, 3) increased ammonification (the release of ammonia) and trace gas

emissions, 4) decreased root biomass, 5) reduced soil fertility (the results of increased cation leaching), 6) increased nitrification (conversion of ammonia to nitrate during decay of litter and soil organic matter), and 7) nitrate leaching resulting in increased nitrate and aluminum concentrations in streams, and decreased water quality (Aber et al., 1989). In addition, studies suggest that during nitrogen saturation, soil microbial communities change from predominantly fungal (mycorrhizal) communities to those dominated by bacteria (Aber et al., 1998). Though the growth of most forests in the U.S. has been and continues to be limited by the nitrogen supply, some U.S. forests are now showing severe symptoms of nitrogen saturation, including high-elevation, non-aggrading spruce-fir ecosystems in the Appalachian Mountains, as well as in the eastern hardwood watersheds at Fernow Experimental Forest near Parsons, West Virginia. Mixed conifer forests and chaparral watersheds with high smog exposure in the Los Angeles Air Basin also are nitrogen saturated and exhibit the highest stream water NO_3^- concentrations for wildlands in North America (Bytnerowicz and Fenn, 1996; Fenn et al., 1998; CD, pp. 4-42 to 4-43). The impact of increasing nitrogen inputs on the nitrogen cycle and forests, wetlands, and aquatic ecosystems is discussed in detail elsewhere (EPA,1993, 1997a; Garner, 1994; World Health Organization, 1997). Understanding the variability in forest ecosystem response to nitrogen input is essential in assessing pollution-related impacts (CD, p. 4-49).

As noted above, sulfur is another essential plant nutrient, the most important source of which for plants is sulfate taken up by the roots, even though plants can also utilize atmospheric SO_2. Atmospheric deposition of sulfate to the soils, therefore, is an important component of the sulfur cycle. The biochemical relationship between sulfur and nitrogen in plant proteins indicates that neither element can be assessed adequately without reference to the other. Nitrogen uptake in forests may be loosely regulated by sulfur availability, but sulfate additions in excess of needs do not necessarily lead to injury. (CD, pp. 4-51 to 4-52).

The nutritional needs of plants also include a suite of other essential minerals such as calcium (Ca), magnesium (Mg) and potassium (K). Soil acidification and its effects result from the deposition of nitrate ($N0_3^-$) and sulfate (SO_4^{-2}) and the associated hydrogen (H^+) ion. The introduction of H^+ by atmospheric deposition or by internal processes will directly impact the fluxes of base cations such as Ca, K, and Mg via cation exchange or weathering processes.

Therefore, soil leaching is often of major importance in cation cycles, and many forest ecosystems show a net loss of base cations. In aluminum-rich soils, acid deposition, by lowering the pH, can increase aluminum concentrations in soil water through dissolution and ion-exchange processes. There is abundant evidence that aluminum is toxic to plants, and it is believed that the toxic effect of aluminum on forest trees could be due to its interference with Ca uptake. Once it enters the forest tree roots, aluminum accumulates in root tissue. Because calcium plays a major role in cell membrane integrity and cell wall structure, reductions in Ca uptake suppresses cambial growth, reduces the rate of wood formation, decreases the amount of functional sapwood and live crown and predisposes trees to disease and injury from stress agents when the functional sapwood becomes less than 25% of cross sectional stem area. There are large variations in Al sensitivity among ecotypes, between and within species due to differences in nutritional demands and physiological status, which are related to age and climate, which change over time (CD, pp. 4-53 to 4-60).

The Integrated Forest Study (IFS) (Johnson and Lindberg, 1992) has characterized the complexity and variability of ecosystem response to atmospheric inputs and provided the most extensive data set available on the effects of atmospheric deposition, including particle deposition, on the cycling of elements in forest ecosystems. The IFS project concluded that acidic deposition is having a significant, often overwhelming effect on both nutrient cycling and cation leaching from the soils in most of the forest ecosystems studied, though the nature of the effects varies from one location to another. It appears that particle deposition has a greater effect on base cation inputs to soils than on base cation losses associated with inputs of sulfur, nitrogen, and H^+. These inputs of base cations have considerable significance, not only to the base cation status of these ecosystems, but also to the potential of incoming precipitation to acidify or alkalize the soils in these ecosystems. However, these net losses or gains of base cations must be placed in the context of the existing soil pool size of exchangeable base cations. The actual rates, directions, and magnitudes of changes that may occur in soils (if any) will depend on rates of inputs from weathering, vegetation outputs, as well as deposition and leaching. In some cases, sites identified as sensitive have large stores of weatherable minerals, while other soils, with smaller stores of weatherable minerals but larger exchangeable cation reserves, are considered less sensitive. In

addition, atmospheric deposition may have significantly affected the nutrient status of some IFS sites through the mobilization of Al. However, the connection between Al mobilization and forest response is still not clear and warrants further study (CD, pp. 4-62 to 4-72).

Trace Elements. Some trace elements deposited directly onto vegetative surfaces can be toxic to the populations of fungi and other microorganisms living on the leaves. Since these organisms play an important role in leaf decomposition after litterfall, changes in these communities can affect the rate of litter decomposition and subsequently nutrient availability for vegetation. Alternatively, trace elements can be absorbed and bound in the leaf tissue, which has also been shown to have a depressing effect on the rates of litter decomposition. Heavy metals deposited from the atmosphere to forests accumulate either in the top, richly organic layer of the forest floor or in the soil layers immediately beneath it, areas where the activity of plant roots and soil organisms is greatest. Because copper, nickel, zinc, cadmium, cobalt and lead compounds can all be toxic to roots and soil organisms, these heavy metals change the litter decomposition processes which influence the availability of essential soil nutrients, ultimately interfering with ecosystem nutrient cycling. Therefore, any effects on structure and function of an ecosystem are likely to occur through the soil and litter. A number of toxic effects of metals on soil microbes have been documented. For example, cadmium was observed to decrease and prolong logarithmic rates of microbial increase, to reduce microbial respiration and fungal spore formation and germination, to inhibit bacterial transformation, and to induce abnormal morphologies. Additionally, the effects of metals on the symbiotic activity of fungi, bacteria, and actinomycetes to plant roots can vary from host to host (Gildon and Tinker, 1983). Alternately, symbiotic associations of mycorrhizal fungi with plants may also provide some additional degree of tolerance to metals (CD, pp. 4-77 to 4-81).

There is some evidence that invertebrates inhabiting soil litter do accumulate metals. Earthworms from roadsides were shown to contain elevated concentrations of cadmium, nickel, lead, and zinc, though interference with earthworm activity was not cited. A study of the accumulation of these same metals in earthworms suggested that cadmium and zinc were concentrated, but not lead. It has further been shown that when soils are acidic, earthworm abundance decreases and bioaccumulation of metals from the soil may increase exponentially with

decreasing pH. Thus, organisms that feed on earthworms from soils with elevated concentrations of lead and zinc for extended periods would be expected to accumulate these metals to toxic levels. Biological accumulation of metals through the plant-herbivore and litter-detritivore chains can occur. Studies indicate that heavy metal deposition onto the soil, via food chain accumulation, can cause excess levels and toxic effects in certain animals (CD, pp. 4-78 to 4-81).

Organics. At the ecosystem level, some organic chemicals are of concern because they may reach toxic levels in both animal and human food chains. Of particular ecological and public concern are the polychlorinated hydrocarbons, such as the dioxins. As discussed above, wet and dry particle deposition are the most important pathways for the accumulation of these more highly chlorinated congeners in vegetation. Though not studied extensively, biodegradation probably does not occur since these compounds are found primarily in the lipophilic cuticle and are very resistant to microbial degradation. Therefore, the grass-cattle-milk/beef pathway is a critical one for humans since exposure often comes from ingestion of animal fat from fish, meat and dairy products. Alternatively, feed contaminated with soil containing the pollutant can be another source of exposure of beef and dairy cattle as well as chickens. Likewise in natural ecosystems, these chemicals tend to bioaccumulate up the food chain. Actions taken by EPA (under the authority of Section 112 of the CAA) and others to evaluate and control sources of Great Waters pollutants of concern appear to have positively affected trends in pollutant concentrations measured in air, sediment, and biota. (CD, pp. 4-30 to 4-32).

5.4.3 Summary

The draft CD presents evidence of effects on vegetation and ecosystems from ambient PM, both in the U.S. and Europe, including in particular effects related to nitrate and acidic deposition. Based on available evidence, the draft CD concludes that "atmospheric PM at levels currently found in the United States has the potential to alter ecosystem structure and function in ways that may reduce their ability to meet societal needs." (CD, p. 4-84). However, the available information does not yet provide the basis to characterize quantitatively the complex relationships between observed adverse effects on vegetation and ecosystems in various locations across the U.S. and levels of PM in the ambient air, due in part to the role that location-specific

environmental factors play, even in determining whether PM deposition occurring in a given location represents a beneficial or an adverse effect. Thus, while evidence of PM-related effects clearly exists, there is insufficient information available at this time to serve as a basis for a national PM air quality standard, defined in terms of concentrations of fine- and/or coarse-fraction particles in the ambient air, specifically selected to protect against adverse effects on vegetation and ecosystems.

5.5 EFFECTS ON SOLAR RADIATION AND GLOBAL CLIMATE CHANGE

The extensive international research and assessment efforts into stratospheric ozone depletion and global climate change provide evidence that atmospheric particles play important roles in two key types of atmospheric processes: 1) alterations in the amount of solar radiation in the ultraviolet range (especially UV-B radiation) penetrating through the earth's atmosphere and reaching its surface, where it can exert a variety of effects on human health, plant and animal biota, and other environmental components; and 2) alterations in the amount of solar radiation in the visible range being transmitted through the earth's atmosphere and either being reflected back into space or absorbed (as well as a lessor role in absorbing infrared radiation emitted by the earth's surface), which enhance heating of the earth's surface and lower atmosphere and lead to consequent "global warming" impacts on human health and the environment (CD, p. 4-129). Information on the role of atmospheric particles in these atmospheric processes is summarized above in Chapter 2 (Section 2.9). Based on information in Chapter 4 of the draft CD, the effects on human health and the environment associated with such atmospheric processes are summarized below, in conjunction with consideration of the potential indirect impacts on human health and the environment that may be a consequence of radiative and climatic changes attributable to changes in ambient PM.

5.5.1 Alterations in Solar UV-B Radiation and Potential Human Health and Environmental Impacts

This section briefly summarizes information on the health and environmental effects associated with UV-B radiation exposure and considers the potential impacts that may result from

changes in UV-B radiation penetration to the earth's surface attributable to changes in ambient PM. The main types of effects associated with exposure to UV-B radiation include direct effects on human health and agricultural and ecological systems, indirect effects on human health and ecosystems, and effects on materials. The study of these effects has been driven by international concern over potentially serious increases in the amount of solar UV-B radiation reaching the earth's surface due to the depletion of the stratospheric ozone layer by the release of various man-made ozone-depleting substances. Extensive qualitative and quantitative characterizations of these global effects attributable to projections of stratospheric ozone depletion have been periodically assessed in studies carried out under WMO and UNEP auspices, with the most recent projections being published by UNEP (1998).

Direct human health effects of UV-B radiation exposure include: skin damage (sunburn) leading to more rapid aging and increased incidence of skin cancer; effects on the eyes, including retinal damage and increased cataract formation possibly leading to blindness; and suppression of some immune system components, contributing to skin cancer induction and possibly increasing susceptibility to certain infectious diseases and/or decreasing effectiveness of vaccinations. Direct environmental effects include damage to terrestrial plants, leading to possible reduced yields of some major food crops and commercially important tress, as well as to biodiversity shifts in natural terrestrial ecosystems; and adverse effects on aquatic life, including reductions in important components of marine food chains as well as other aquatic ecosystem shifts. Indirect health and environmental effects are primarily those mediated through increased tropospheric ozone formation and consequent ozone-related health and environmental impacts. Effects on materials include accelerated polymer weathering and other effects on man-made materials and cultural artifacts. In addition, there are emerging complex issues regarding interactions and feedbacks between climate change and changes in terrestrial and marine biogeochemical cycles due to increased UV-B radiation penetration.

The various assessments of these effects that have been conducted consistently note that the modeled projections quantitatively relating changes in UV-B radiation (attributable to stratospheric ozone depletion) to changes in health and environmental effects are subject to considerable uncertainty, with the role of atmospheric particles being one of numerous

complicating factors. Taking into account the complex interactions between ambient particles and UV-B radiation transmission through the lower atmosphere, the CD concludes that any effort to quantify projected indirect effects of variations in atmospheric PM on human health or the environment due to particle impacts on transmission of solar UV-B radiation would require location-specific evaluations that take into account the composition, concentration, and internal structure of the particles; temporal variations in atmospheric mixing heights and depths of layers containing the particles; and consequent impacts on surface level exposures of humans, ecosystem constituents, or man-made materials (CD, page 4-137).

At present, models are not available to take such complex factors into account, nor is sufficient data available to characterize input variables that would be necessary for any such modeling. The CD concludes, however, that the outcome of such modeling efforts would likely vary from location to location, even as to the direction of changes in the levels of exposures to UV-B radiation, due to location-specific changes in ambient PM concentrations and/or composition (CD, p. 4-137). Beyond considering just average levels of exposures to UV-B radiation in general, the CD notes that ambient PM can affect the directional characteristics of UV-B radiation scattering at ground-level, and thus its biological effectiveness. Also, ambient PM can affect not only biologically damaging UV-B radiation, but can also reduce the ground-level ratio of photorepairing UV-A radiation to UV-B radiation. Further, the CD notes that ambient PM deposition is a major source of PAH in certain water bodies, which can enhance the adverse effects of solar UV-B radiation on aquatic organisms, such that the net effect of ambient PM in some locations may be to increase UV-B radiation-related biological damage to certain aquatic and terrestrial organisms.

5.5.2 Global Climate Change and Potential Human Health and Environmental Impacts

This section briefly summarizes information on the health and environmental vulnerabilities associated with global warming and climate change, and considers the potential impacts that may result from such climatic changes attributable to changes in ambient PM. In general, a number of sectors are seen as vulnerable to climatic change resulting from global warming, including terrestrial and aquatic ecosystems, hydrology and water resources, food and fiber production,

coastal systems, and human health (Intergovernmental Panel on Climate Change, 1998). The study of these vulnerabilities has been driven by international concern over increases in emissions due to man's activities of "greenhouse gases," or their precursors, leading to consequent global warming and climate change. These gases include especially carbon dioxide, nitrous oxide, methane, chlorofluorocarbons, and tropospheric ozone. The presence of ambient PM is one of numerous factors that plays a role in the extremely complex assessment of such climatic changes. The processes involved in global warming and its likely consequent effects have been extensively reviewed, with all assessments and summaries emphasizing the extreme complexity associated with such assessment. Despite the inherent complexity and uncertainties in these global-scale assessments, all typically agree that some global warming has occurred and will continue to occur during the coming decades. Further, the impacts are generally projected to be highly variable across geographic regions, with the potential for both substantial damage in some sectors, or, conversely, the potential for some beneficial outcomes. The most recent report on possible global climate change impacts on various areas in the U.S. is based on assessments now being conducted by the U.S. Global Change Research Program (USGCRG, 2000), summarized in the CD (Appendix 4D).

Potential effects of global warming and climate change on both the environment and human health in the U.S. are summarized in the CD (Section 4.5.2). The most vulnerable environmental sectors and regions in the continental U.S. include long-lived natural forest ecosystems in the East and interior West; water resources in the southern plains; agriculture in the Southeast and southern plains; northern ecosystems and habitats; estuary beaches in developed areas; and low-latitude cool and cold water fisheries. On the other hand, other sectors or subregions may benefit, including west coast coniferous forests; some western rangelands; reduced energy costs for heating in northern latitudes; reduced road salting and snow-clearance costs; longer open-water seasons in northern channels and ports; and agriculture in northern latitudes, the interior West, and the west coast. Both adverse and beneficial environmental effects are projected for Alaska, with possible major declines or loss of some sensitive species occurring in parallel with possible opening of ice-bound transportation routes or expanded agriculture.

With regard to effects on human health, mainly deleterious direct and indirect effects are projected to be associated with global warming and climate change. Such direct health effects include increased mortality linked to temperature extremes (both high and low) and increases in the incidence and spread of vector-borne infectious diseases (e.g., Lyme disease, malaria). Indirect health effects include effects secondary to sea-level rise (e.g., changes in the habitats of mosquitos and other disease vectors) and those secondary to increased tropospheric air pollution (e.g., respiratory effects associated with exposure to ground-level ozone).

The CD (p. 4-154) notes that observational evidence for the climatic effects of ambient particles is sparse. Further, any effort to model the relationship between changes in ambient PM and direct climatic effects would be hindered by a lack of knowledge of ambient particle characteristics including vertical and horizontal variability, size distribution, chemical composition and the distribution of components within individual particles. The CD stresses that the overall radiative effect of particles at a given location is not simply determined by the sum of effects caused by individual classes of particles because of interactions between particles and atmospheric gases. Further, estimation of indirect particle effects are subject to even much greater uncertainties. The CD concludes that, although on a global scale atmospheric particles likely exert an overall net effect of slowing global warming, much uncertainty would be associated with any future efforts aimed at projecting the net effect on global warming processes, resulting climate change, and any consequent human health or environmental effects, due to location-specific changes in emissions of particles or their gaseous precursors (CD, page 4-155).

5.5.3 Summary

A number of assessments of the factors affecting the penetration of solar UV-B radiation to the earth's surface and of the factors affecting global warming and climate change clearly recognize ambient PM as playing various roles in these processes. These assessments, however, have focused on global- and regional-scale impacts, allowing for generalized assumptions to take the place of specific, but unavailable, information on local-scale atmospheric parameters and characteristics of the distribution of particles present in the ambient air. As such, the available information provides no basis for estimating how localized changes in the temporal, spatial, and

composition patterns of ambient PM, likely to occur as a result of expected future emissions of particles and their precursor gases across the U.S., would affect local, regional, or global changes in UV-B radiation penetration and scattering or global warming – even the direction of such effects on a local scale remains uncertain. Moreover, similar concentrations of different particle components can produce opposite net effects. It follows, therefore, that there is insufficient information available to project the extent to which, or even whether, such location-specific changes in ambient PM would indirectly affect human health or the environment secondary to potential changes in UV-B radiation and global warming.

Based on currently available information, the indirect effects of ambient PM, secondary to potential changes in UV-B radiation and global warming, can play no quantitative role in considering whether any revisions of the primary or secondary PM NAAQS are appropriate at this time. Even qualitatively, the available information is very limited in the extent to which it can help inform an assessment of the overall weight of evidence in an assessment of the net health and environmental effects of PM in the ambient air, considering both its direct effects (e.g., inhalation-related health effects) and indirect effects mediated by other routes of exposure and environmental factors (e.g., dermal exposure to UV-B radiation).

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APPENDIX A TABLE 1. ESTIMATED INCREASED MORTALITY PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Total (nonaccidental) Mortality				
Ito and Thurston, 1996 *Chicago, IL*	*2.47 (1.26, 3.69)*	---	---	*PM_{10} 38 (max 128)*
Kinney et al., 1995 *Los Angeles, CA*	*2.47 (-0.17, 5.18)*	---	---	*PM_{10} 58 (15, 177)*
Pope et al., 1992 *Utah Valley, UT*	*7.63 (4.41, 10.95)*	---	---	*PM_{10} 47 (11, 297)*
Schwartz, 1993 *Birmingham, AL*	*5.36 (1.16, 9.73)*	---	---	*PM_{10} 48 (21, 80)*
Schwartz et al., 1996 *Boston, MA*	*6.15 (3.56, 8.80)*	*5.59 (3.80, 7.42)*	*0.51 (-1.73, 2.78)*	*PM_{10} 24.5 (SD 12.8)* *$PM_{2.5}$ 15.7 (SD 9.2)* *$PM_{10-2.5}$ 8.8 (SD 7.0)*
Schwartz et al., 1996 *Knoxville, TN*	*4.58 (0.27, 9.08)*	*3.54 (0.52, 6.65)*	*2.52 (-1.46, 6.66)*	*PM_{10} 32.0 (SD 14.5)* *$PM_{2.5}$ 20.8 (SD 9.6)* *$PM_{10-2.5}$ 11.2 (SD 7.4)*
Schwartz et al., 1996 *St. Louis, MO*	*3.04 (0.76, 5.37)*	*2.77 (1.13, 4.44)*	*0.50 (-1.73, 2.78)*	*PM_{10} 30.6 (SD 16.2)* *$PM_{2.5}$ 18.7 (SD 10.5)* *$PM_{10-2.5}$ 11.9 (SD 8.5)*
Schwartz et al., 1996 *Steubenville, OH*	*4.58 (0.76, 8.54)*	*2.52 (-0.24, 5.35)*	*6.11 (1.30, 11.15)*	*PM_{10} 45.6 (SD 32.3)* *$PM_{2.5}$ 29.6 (SD 21.9)* *$PM_{10-2.5}$ 16.1 (SD 13.0)*
Schwartz et al., 1996 *Portage, WI*	*3.55 (-1.71, 9.09)*	*3.03 (-0.84, 7.05)*	*1.25 (-3.06, 5.76)*	*PM_{10} 17.8 (SD 11.7)* *$PM_{2.5}$ 11.2 (SD 7.8)* *$PM_{10-2.5}$ 6.6 (SD 6.8)*

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1996 Topeka, KS	*-2.48 (-9.33, 4.90)*	*2.01 (-4.83, 9.35)*	*-3.22 (-7.89, 1.69)*	*PM_{10} 26.7 (SD 16.1) $PM_{2.5}$ 12.2 (SD 7.4) $PM_{10-2.5}$ 14.5 (SD 12.2)*
Schwartz et al., 1996 6 Cities, Overall	*4.06 (2.53, 5.62)*	*3.79 (2.77, 4.82)*	*1.00 (-0.37, 2.40)*	*PM_{10} means 17.8-45.6 $PM_{2.5}$ means 11.2-29.6 $PM_{10-2.5}$ means 6.6-16.1*
Styer et al., 1995 Chicago, IL	*4.08 (0.08, 8.24)*	---	---	*PM_{10} 37 (4, 365)*
Samet et al., 2000a,b 90 Largest U.S. Cities	2.27 (0.10, 4.48)	---	---	PM_{10} mean range 15.3-52.0
Samet et al., 2000c 20 Largest U.S. Cities	2.58 (0.41, 4.79)	---	---	PM_{10} mean range 23.8-46.0
Dominici et al., 2000 20 Largest U.S. Cities	1.91 (-0.41, 4.30)	---	---	PM_{10} mean range 23.8-52.0
Schwartz, 2000a 10 U.S. cities	3.40 (2.65, 4.14)	---	---	PM_{10} mean range 27.1-40.6
Braga et al., 2000 5 U.S. cities	4.3 (3.0, 5.6)	---	---	PM_{10} mean range 28-37
Burnett et al., 1998 Toronto, CAN	3.46 (1.74, 5.21)	4.79 (3.26, 6.34)	---	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (8, 90)
Burnett et al., 2000 8 Canadian Cities	3.51 (1.04, 6.04)	3.03 (1.10, 4.99)	1.82 (-0.72, 4.43)	PM_{10} 25.9 (max 121) $PM_{2.5}$ 13.3 (max 86) $PM_{10-2.5}$ 12.9 (max 99)
Chock et al., 2000 Pittsburgh, PA		<75 years 2.6 (2.0, 7.3) >75 years 1.5 (-3.0, 6.3)	<75 years 0.7 (-1.7, 3.7) >75 years 1.3 (-1.3, 3.8)	NR
Clyde et al., 2000 Phoenix, AZ	6 (>0, 11)	---	---	PM_{10} mean 45.4

Reference, Study Location *	% increase (95% CI) per 50 $\mu g/m^3$ PM_{10} Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Fairley, 1999 Santa Clara County, CA	8 ($p<0.05$)	8 ($p<0.01$)	2 ($p>0.05$)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Gamble, 1998 Dallas, TX	-3.56 (-12.73, 6.58)	---	---	PM_{10} 24.5 (11, 86)
Goldberg et al., 2000 Montreal, CAN	---	5.81 (3.36, 8.32)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	12.33 (2.50, 23.11)	1.54 (0.3, 2.74) (15 ug/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) nmol/m^3
Klemm and Mason, 2000 Atlanta, GA	---	4.8 (-3.2, 13.4)	1.4 (-11.3, 15.9)	$PM_{2.5}$ 19.9 (1.0, 54.8) $PM_{10-2.5}$ 10.1 (0.2, 39.5)
Klemm et al., 2000 Six City reanalysis - St. Louis	2.02 (-0.24, 4.33)	2.01 (0.51, 3.54)	0.25 (-1.98, 2.53)	PM_{10} 30.6 (SD 16.2) $PM_{2.5}$ 18.7 (SD 10.5) $PM_{10-2.5}$ 11.9 (SD 8.5)
Klemm et al., 2000 Six City reanalysis - Steubenville	3.04 (-1.23, 7.48)	1.51 (-1.60, 4.71)	4.82 (4.04, 5.61)	PM_{10} 45.6 (SD 32.3) $PM_{2.5}$ 29.6 (SD 21.9) $PM_{10-2.5}$ 16.1 (SD 13.0)
Klemm et al., 2000 Six City reanalysis - Topeka	-3.45 (-11.37, 5.17)	1.51 (-6.48, 10.18)	-3.71 (-9.17, 2.08)	PM_{10} 26.7 (SD 16.1) $PM_{2.5}$ 12.2 (SD 7.4) $PM_{10-2.5}$ 14.5 (SD 12.2)
Klemm et al., 2000 Six City reanalysis - overall	4.06 (2.78, 5.36)	3.28 (2.27, 4.31)	1.00 (-0.37, 2.40)	PM_{10} means 17.8-45.6 $PM_{2.5}$ means 11.2-29.6 $PM_{10-2.5}$ means 6.6-16.1
Klemm et al., 2000 Six City reanalysis - Knoxville	7.20 (2.29, 12.34)	4.82 (1.40, 8.35)	4.05 (-0.46, 8.76)	PM_{10} 32.0 (SD 14.5) $PM_{2.5}$ 20.8 (SD 9.6) $PM_{10-2.5}$ 11.2 (SD 7.4)

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Klemm et al., 2000 Six City reanalysis - Boston	6.15 (3.56, 8.80)	5.33 (3.54, 7.15)	1.25 (-1.11, 3.68)	PM_{10} 24.5 (SD 12.8) $PM_{2.5}$ 15.7 (SD 9.2) $PM_{10-2.5}$ 8.8 (SD 7.0)
Klemm et al., 2000 Six City reanalysis - Madison	2.02 (-3.42, 7.76)	2.27 (-1.83, 6.54)	0.25 (-4.51, 5.25)	PM_{10} 17.8 (SD 11.7) $PM_{2.5}$ 11.2 (SD 7.8) $PM_{10-2.5}$ 6.6 (SD 6.8)
Laden et al., 2000 Six City reanalysis	---	4.05 (2.78, 5.34) overall -5.65 (-13.74, 3.19) crustal 8.72 (4.22, 13.41) mobile 2.77 (0.64, 4.95) coal	---	$PM_{2.5}$ same as Six City
Levy et al., 1998 King Co., WA	7.2 (-6.3, 22.8)	1.76 (-3.53, 7.34)	---	PM_{10} 29.8 (6.0, 123.0) PM_{1} 28.7 (16.3, 92.2)
Lipfert et al., 2000 Philadelphia, PA	5.99 (p>0.055)	4.21 (p<0.055)	5.07 (p>0.055)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	4.41 (-0.98, 10.10)	3.10 (-0.63, 6.98)	3.96 (-1.22, 9.42)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (5%, 95%)
Mar et al., 2000 Phoenix, AZ	5.44 (0.06, 11.12)	5.98 (-1.34, 13.85)	2.97 (-0.50, 6.56)	PM_{10} 46.5 (5, 213) $PM_{2.5}$ 13.0 (0, 42) $PM_{10-2.5}$ 33.5 (5, 187)
Moolgavkar, 2000a Los Angeles, CA	1.25 (p<0.05, from figure)	0.6 (p>0.05, from figure)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL	1.25 (p<0.05, from figure)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Maricopa Co., AZ	3 (p<0.05, from figure)	---	---	PM_{10} median 41 (9, 252)

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	0.28 (-0.61, 1.17)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Ostro et al., 1999 Coachella Valley, CA	4.60 (0.58, 8.79)	---	---	PM_{10} 56.8 (38, 417)
Ostro et al., 2000 Coachella Valley, CA	2.01 (-0.99, 5.10)	11.8 (1.3, 23.4)	0.7 (-0.8, 2.3)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Pope et al., 1999 Ogden, UT	12.02 (4.49, 20.99)	---	---	PM_{10} 32.1 (4, 182)
Pope et al., 1999 Salt Lake City, UT	2.33 (0.05, 4.66)	---	---	PM_{10} 41.2 (7, 441)
Pope et al., 1999 Provo/Orem, UT	1.87 (-2.15, 6.04)	---	---	PM_{10} 38.4 (1, 317)
Schwartz, 2000c Boston, MA	---	5.33 (1.81, 8.98)	---	$PM_{2.5}$ 15.6 (±9.2)
Schwartz and Zanobetti, 2000 Chicago, IL	4.53 (3.11, 5.96)	---	---	PM_{10} median 36
Tsai et al., 2000 Newark, NJ	5.65 (4.62, 6.70)	4.34 (2.82, 5.89)	---	PM_{15} 55 (SD 6.5) $PM_{2.5}$ 42.1 (SD 22.0)
Tsai et al., 2000 Camden, NJ	11.07 (0.70, 22.51)	5.65 (0.11, 11.51)	---	PM_{15} 47.0 (SD 20.9) $PM_{2.5}$ 39.9 (SD 18.0)
Tsai et al., 2000 Elizabeth, NJ	-4.88 (-17.88, 10.19)	1.77 (-5.44, 9.53)	---	PM_{15} 47.5 (SD 18.8) $PM_{2.5}$ 37.1 (SD 19.8)
Cause-Specific Mortality				
Cardiorespiratory:				

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Samet et al., 2000c 20 Largest U.S. Cities	3.45 (1.01, 5.94)	---	---	PM_{10} means 15.3-46.0
Tsai et al., 2000 Newark, NJ	7.79 (3.65, 12.10)	5.13 (3.09, 7.21)	---	PM_{15} 55 (SD 6.5) $PM_{2.5}$ 42.1 (SD 22.0)
Tsai et al., 2000 Camden, NJ	15.03 (4.29, 26.87)	6.18 (0.61, 12.06)	---	PM_{15} 47.0 (SD 20.9) $PM_{2.5}$ 39.9 (SD 18.0)
Tsai et al., 2000 Elizabeth, NJ	3.05 (-11.04, 19.36)	2.28 (-4.97, 10.07)	---	PM_{15} 47.5 (SD 18.8) $PM_{2.5}$ 37.1 (SD 19.8)
Total Cardiovascular:				
Ito and Thurston, 1996 *Chicago, IL*	*1.49 (-0.72, 3.74)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*9.36 (1.91, 17.36)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA	9 ($p<0.05$)	6.2 ($p>0.05$)	3 ($p>0.05$)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Goldberg et al., 2000 Montreal, CAN	---	3.48 (-0.16, 7.26)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	6.86 (-1.28, 15.66)	1.54 (-1.14, 4.28) (15 ug/m³ $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) nmol/m³
Lipfert et al., 2000 Philadelphia, PA (7-county area)	6.92 ($p<0.055$)	10.26 ($p<0.055$)	7.57 ($p>0.055$)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	6.86 (-1.28, 15.66)	3.17 (-2.29, 8.94)	7.82 (0.03, 16.23)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Mar et al., 2000 Phoenix, AZ	9.86 (1.91, 18.42)	18.68 (5.72, 33.23)	6.45 (1.42, 11.73)	PM_{10} 46.5 (5, 213) $PM_{2.5}$ 13.0 (0, 42) $PM_{10-2.5}$ 33.5 (5, 187)
Moolgavkar, 2000a Los Angeles, CA	4.47 (1.65, 7.37)	2.59 (0.38, 4.85)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL	2.21 (0.37, 4.09)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Maricopa Co., AZ	8.85 (2.67, 15.39)	---	---	PM_{10} median 41 (9, 252)
Ostro et al., 2000 Coachella Valley, CA	6.09 (2.05, 10.29)	8.56 (-6.35, 25.84)	2.56 (0.60, 4.49)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Ostro et al., 1999 Coachella Valley, CA	8.33 (2.14, 14.9)	---	---	PM_{10} 56.8 (38, 417)
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	0.69 (-0.34, 1.74)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Pope et al., 1999 Salt Lake City, UT	6.50 (2.21, 10.98)	---	---	PM_{10} 41.2 (7, 441)
Pope et al., 1999 Provo/Orem, UT	8.60 (2.40, 15.18)	---	---	PM_{10} 38.4 (1, 317)
Pope et al., 1999 Ogden, UT	1.41 (-8.33, 12.18)	---	---	PM_{10} 32.1 (4, 182)
Coronary Artery Disease:				
Goldberg et al., 2000 Montreal, CAN	---	4.48 (-0.31, 9.51)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Cerebrovascular:				

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Moolgavkar, 2000a Cook Co., IL	3.27 (-0.12, 6.77)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA	2.92 (-2.27, 8.39)	3.61 (-0.57, 7.97)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Maricopa Co., AZ	11.09 (0.54, 22.75)	---	---	PM_{10} median 41 (9, 252)
Total Respiratory:				
Ito and Thurston, 1996 *Chicago, IL*	*6.77 (1.97, 11.79)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*19.78 (3.51, 38.61)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA	11 (p>0.05)	11.5 (p>0.05)	16 (p>0.05)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Goldberg et al., 2000 Montreal, CAN	---	21.6 (13.0, 31.0)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	17.89 (-14.87, 63.25)	8.16 (4.18, 12.30) (15 ug/m³ $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) nmol/m³
Lipfert et al., 2000 Philadelphia, PA (7-county area)	-3.17 (p>0.055)	0.66 (p>0.055)	-12.72 (p>0.055)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	7.84 (-10.18, 29.47)	2.28 (-10.31, 16.63)	7.41 (-9.07, 26.87)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)
Ostro et al., 1999 Coachella Valley, CA	13.88 (3.25, 25.61)	---	---	PM_{10} 56.8 (38, 417)

Reference, Study Location *	% increase (95% CI) per 50 μg/m³ PM_{10} Increase	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Ostro et al., 2000 Coachella Valley, CA	-1.99 (-11.41, 8.44)	-13.28 (-43.05, 32.06)	-1.27 (-6.24, 3.95)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	2.08 (-0.35, 4.51)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Pope et al., 1999 Ogden, UT	23.80 (2.77, 49.14)	---	---	PM_{10} 32.1 (4, 182)
Pope et al., 1999 Provo/Orem, UT	2.22 (-9.83, 15.89)	---	---	PM_{10} 38.4 (1, 317)
Pope et al., 1999 Salt Lake City, UT	8.17 (-0.97, 18.14)	---	---	PM_{10} 41.2 (7, 441)
COPD:				
Moolgavkar, 2000a Cook Co., IL	5.39 (0.30, 10.74)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA	5.91 (-1.64, 14.03)	2.67 (-3.38, 9.10)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Maricopa Co., AZ	8.08 (-4.58, 22.41)	---	---	PM_{10} median 41 (9, 252)

* Studies in italics available in 1996 CD

** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 2. ESTIMATED RESPIRATORY MORBIDITY EFFECTS PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Reference, Study Location*	% increase (95% CI) per 50 $\mu g/m^3$ PM_{10} Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Increased Admission to Hospital or Emergency Room				
Total Respiratory:				
Thurston et al., 1994 Toronto, Canada	*23.26 (2.03, 44.49)*	*15.00 (1.97, 28.03)*	*22.25 (-9.53, 54.03)*	*PM_{10} 29.5-38.8 (max 96.0) $PM_{2.5}$ 15.8-22.3 (max 66.0) $PM_{10-2.5}$ 12.7-16.5 (max 33.0)*
Schwartz, 1995 New Haven, CT	*6.00 (-0.28, 12.68)*	---	---	*PM_{10} 41 (19-67)****
Schwartz, 1995 Tacoma, WA	*10.00 (3.21, 17.23)*	---	---	*PM_{10} 37 (14-67)****
Schwartz et al., 1996 Spokane, WA	*8.50 (3.61, 13.62)*	---	---	*PM_{10} 46 (16-83)****
Schwartz et al., 1996 Cleveland, OH	*5.83 (0.54, 11.40)*	---	---	*PM_{10} 43 (19-72)****
Gwynn et al., 2000 Buffalo, NY	17.27 (0.61, 36.68)	8.16 (4.18, 12.30) (15 $\mu g/m^3$ $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) nmol/m^3
Linn et al., 2000 Los Angeles, CA (>29 years)	2.89 (1.09, 4.72)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	8.72 (4.59, 13.01) (COPD + pneumonia)	---	---	PM_{10} 34.0 (17, 55)
Moolgavkar et al., 1997 Birmingham, AL (>65 years)	1.51 (-1.43, 4.54) (COPD + pneumonia)	---	---	PM_{10} 43.4 (18.5, 74.1)
Schwartz et al., 1996 Cleveland, OH (>65 years)	5.83 (0.54, 11.40)	---	---	PM_{10} 43

Lumley and Heagerty, 1999 King County, WA (all ages)	---	5.91 (1.10, 10.97)	---	PM_1 NR
Burnett et al., 1997 Toronto, CAN (all ages)	10.93 (4.53, 17.72)	8.61 (3.39, 14.08)	12.71 (5.33, 20.74)	PM_{10} 28.1 (4, 102) $PM_{2.5}$ 16.8 (1, 66) $PM_{10-2.5}$ 11.6 (1, 56)
Delfino et al., 1997 Montreal, CAN (>64 years)	36.62 (10.02, 63.21)	23.88 (4.94, 42.83)	---	summer 93 PM_{10} 21.7 (max 51) $PM_{2.5}$ 12.2 (max 31)
Delfino et al., 1998 Montreal, CAN (>64 years)	---	13.17 (-0.22, 26.57)	---	$PM_{2.5}$ 18.6 (SD 9.3)
Stieb et al., 2000 St. John, CAN (all ages)	8.8 (1.8, 16.4)	5.69 (0.61, 11.03)	---	summer 93 PM_{10}14.0 (max 70.3) $PM_{2.5}$ 8.5 (max 53.2)
Pneumonia:				
Schwartz 1994b *Birmingham, AL*	*9.09 (3.51, 14.97)*	---	---	*PM_{10} 45 (19-77)****
Schwartz 1994a *Detroit, MI*	*5.92 (1.95, 10.05)*	---	---	*PM_{10} 48 (22-82)****
Schwartz 1994c *Minnesota/St. Paul, MN*	*8.17 (1.22, 15.59)*	---	---	*PM_{10} 36 (18-58)****
Schwartz et al., 1996 *Spokane, WA*	*5.30 (-1.51, 12.58)*	---	---	*PM_{10} 46 (16-83)****
Samet et al., 2000 14 U.S. Cities (>65 years)	10.3 (8.5, 12.1)	---	---	PM_{10} means 24.4-45.3
Lippmann et al., 2000 Detroit, MI (>65 years)	21.4 (8.2, 36.3)	12.5 (3.7, 22.1)	11.9 (0.7, 24.4)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	3.5 (-0.5, 7.7)	---	---	PM_{10} 34 (17, 55)
Respiratory infections:				

Burnett et al., 1999 Toronto, CAN (all ages)	14.2 (9.3, 19.3)	10.77 (7.18, 14.47)	9.31 (4.64, 14.18)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
COPD:				
Schwartz 1994c *Minnesota/St. Paul, MN*	*25.30 (9.47, 43.42)*	---	---	*PM_{10} 36 (18-58)****
Schwartz 1994b *Birmingham, AL*	*12.69 (3.81, 22.34)*	---	---	*PM_{10} 45 (19-77)****
Schwartz 1994a *Detroit, MI*	*10.63 (4.41, 17.21)*	---	---	*PM_{10} 48 (22-82)****
Schwartz et al., 1996 *Spokane, WA*	*17.10 (7.85, 27.14)*	---	---	*PM_{10} 46 (16-83)****
Samet et al., 2000 14 U.S. Cities (>65 years)	10.3 (7.7, 13.0)	---	---	PM_{10} means 24.4-45.3
Chen et al., 2000 Reno-Sparks, NV(all ages)	9.4 (2.2, 17.1)	---	---	PM_{10} 36.6 (1.7, 201.3)
Linn et al., 2000 Los Angeles, CA (>29 years)	1.5 (-0.5, 3.5)	---	---	PM_{10} 45.5 (5, 132)
Tolbert et al., 2000a Atlanta, GA (all ages)	-3.5 (33.0, -29.9)	12.44 (-7.89, 37.24)	-23.03 (-50.69, 20.15)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Lippmann et al., 2000 Detroit, MI (>65 years)	9.6 (-5.3, 26.8)	5.49 (-4.72, 16.80)	9.29 (-4.19, 24.66)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	6.9 (-0.6, 15.0)	---	---	PM_{10} 34 (17, 55)
Moolgavkar et al., 2000 King County WA (all ages)	5.1 (0, 10.4)	6.4 (0.9, 12.1)	---	PM_{10} $PM_{2.5}$ 18.1 (3, 96)
Moolgavkar, 2000c Cook Co., IL (>65 years)	2.4 (-0.2, 5.1)	---	---	PM_{10} median 35 (3, 365)

Moolgavkar, 2000c Los Angeles, CA (>65 years)	6.1 (1.1, 11.3)	5.1 (0.9, 9.41)	5.07 (-0.44, 10.90)	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 224, 86) $PM_{10-2.5}$ NR
Moolgavkar, 2000c Maricopa Co., AZ (>65 years)	6.9 (-4.2, 19.3)	---	---	PM_{10} median 41 (9, 252)
Burnett et al., 1999 Toronto, CAN (all ages)	6.90 (1.32, 12.78)	4.78 (-0.17, 9.98)	12.83 (4.93, 21.33)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Asthma:				
Choudbury et al., 1997 Anchorage, AK Medical Visits (all ages)	20.9 (11.8, 30.8)	---	---	PM_{10} 42.5 (1, 565)
Jacobs et al., 1997 Butte County, CA (all ages)	6.11 (p>0.05)	---	---	PM_{10} 34.3 (6.6, 636)
Linn et al., 2000 Los Angeles, CA (>29 years)	1.5 (-2.4, 5.6)	---	---	PM_{10} 45.5 (5, 132)
Lipsett et al., 1997 Santa Clara Co., CA (all ages)	9.1 (2.7, 15.9) (at 41 F and below)	---	---	PM_{10} 61.2 (9, 165)
Los Angeles, CA Nauenberg and Basu, 1999 (all ages)	20.0 (5.3, 35)	---	---	44.8 (SE 17.23)
Norris et al., 1999 Seattle, WA (<18 years)	75.9 (32.9, 132.8)	44.5 (21.7, 71.4)	---	PM_{10} 21.7 (8.0, 69.3) $PM_{2.5}$ (est) 4.8 (1.2, 32.4)
Norris et al., 2000 Seattle, WA (<19 years)	56.2 (10.4, 121.0)			PM_{10} 21.5 (8.0, 69.3)
Norris et al., 2000 Spokane WA (<19 years)	2.4 (-10.9, 17.6)			PM_{10} 27.9 (4.7, 186.4)
Tolbert et al., 2000b Atlanta, GA (<17 years)	13.2 (1.2, 26.7)	---	---	PM_{10} 38.9 (9, 105)

Tolbert et al., 2000a Atlanta, GA (all ages)	18.8 (-8.7, 54.4)	2.27 (-14.79, 22.74)	21.08 (-18.23, 79.29)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Sheppard et al., 1999 Seattle, WA (<65 years)	13.7 (5.5, 22.6)	8.7 (3.3, 14.3)	11.1 (2.8, 20.1)	PM_{10} 31.5 (90% 55) $PM_{2.5}$ 16.7 (90% 32) $PM_{10-2.5}$ 16.2 (90% 29)
Burnett et al., 1999 Toronto, CAN (all ages)	8.9 (3.7, 14.4)	6.44 (2.47, 10.57)	11.05 (5.75, 16.62)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Increased Respiratory Symptoms	Odds Ratio (95% CI) for 50 ug/m³ increase in PM_{10}	Odds Ratio (95% CI) for 25 ug/m³ increase in $PM_{2.5}$	Odds Ratio (95% CI) for 25 ug/m³ increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1994 *6 U.S. cities* *(children, cough)*	*1.39 (1.05, 1.85)*	*1.24 (1.00, 1.54)*	---	*PM_{10} median 30.0 (max 117)* *$PM_{2.5}$ median 18.0 (max 86)*
Schwartz et al., 1994 *6 U.S. cities* *(children, lower respiratory symptoms)*	*2.03 (1.36, 3.04)*	*1.58 (1.18, 2.10)*	---	*PM_{10} median 30.0 (max 117)* *$PM_{2.5}$ median 18.0 (max 86)*
Neas et al., 1995 *Uniontown, PA* *(children, cough)*	---	*2.45 (1.29, 4.64)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Ostro et al., 1991 *Denver, CO* *(adults, cough)*	*1.09 (0.57, 2.10)*	---	---	*PM_{10} 22 (0.5, 73)*
Pope et al., 1991 *Utah Valley, UT* *(lower respiratory symptoms, schoolchildren)*	*1.28 (1.06, 1.56)*	---	---	*PM_{10} 44 (11, 195)*

Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, asthmatic patients)	*1.01 (0.81, 1.27)*	---	---	*PM_{10} 44 (11, 195)*
Neas et al., 1996 State College, PA (children, cough)	NR	1.48 (1.17, 1.88) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, wheeze)	NR	1.59 (0.93, 2.70) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, cold)	NR	1.61 (1.21, 2.17) (0-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Ostro et al., 1995 Los Angeles, CA (children, asthma episode)	1.05 (0.64, 1.73)	---	---	PM_{10} 55.87 (19.63, 101.42)
Ostro et al., 1995 Los Angeles, CA (children, shortness of breath)	1.51 (1.04, 2.17)	---	---	PM_{10} 55.87 (19.63, 101.42)
Schwartz and Neas, 2000 Six Cities reanalysis (children, cough)	---	1.28 (0.98, 1.67)	1.77 (1.23, 2.54)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 Six Cities reanalysis (children, lower respiratory symptoms)	---	1.61 (1.20, 2.16)	1.51 (0.66, 3.43)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR

Vedal et al., 1998 Port Alberni, CAN (children, cough)	1.40 (1.14, 1.73)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, phlegm)	1.40 (1.03, 1.90)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, nose symptoms)	1.22 (1.00, 1.47)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, sore throat)	1.34 (1.06, 1.69)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, wheeze)	1.16 (0.82, 1.63)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, chest tightness)	1.34 (0.86, 2.09)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, dyspnea)	1.05 (0.74, 1.49)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, any symptom)	1.16 (1.00, 1.34)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Decreased Lung Function	Lung Function change (L/min) (95% CI) for 50 ug/m³ increase in PM_{10}	Lung Function change (L/min) (95% CI) for 25 ug/m³ increase in $PM_{2.5}$	Lung Function change (L/min) (95% CI) for 25 ug/m³ increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Neas et al., 1995 Uniontown, PA (children)	---	*-2.58 (-5.33, +0.35)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*

Thurston et al., (1997) Connecticut summer camp (children)	---	PEFR -5.4 (-12.3, 1.5) (15 $\mu g/m^3$ $SO_4^=$)	---	$SO_4^=$ 7.0 (1.1, 26.7)
Naeher et al., 1999 Southwest VA (adult women)	am PEFR -3.65 (-6.79, -0.51) pm PEFR -1.8 (-5.03, 1.43)	am PEFR -1.83 (-3.44, -0.21) pm PEFR -1.05 (-2.77, 0.67)	am PEFR -6.33 (-12.50, -0.15) pm PEFR -2.4 (-8.48, 3.68)	PM_{10} 27.07 (4.89, 69.07) $PM_{2.5}$ 21.62 (3.48, 59.65) $PM_{10-2.5}$ 5.72 (0.00, 19.78)
Neas et al., 1996 State College, PA (children)	---	pm PEFR -0.64 (-1.73, 0.44)	---	$PM_{2.5}$ 23.5 (max 85.8)
Neas et al., 1999 Philadelphia, PA (children)	am PEFR -8.17 (-14.81, -1.56) pm PEFR -1.44 (-7.33, 4.44)	am PEFR -3.29 (-6.64, 0.07) pm PEFR -0.91 (-4.04, 2.21)	am PEFR -4.31 (-11.44, 2.75) pm PEFR 1.88 (-4.75, 8.44)	$PM_{2.5}$ 22.2 (IQR 16.2) $PM_{10-2.5}$ 9.5 (IQR 5.1)
Schwartz and Neas, 2000 Uniontown, PA (reanalysis) (children)	---	pm PEFR -1.52, (-2.80, -0.24)	pm PEFR +1.73 (-2.2, 5.67)	$PM_{2.5}$ 24.5 (max 88.1) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 State College PA (reanalysis) (children)	---	pm PEFR -0.93 (-1.88, 0.01)	pm PEFR -0.28 (-3.45, 2.87)	$PM_{2.5}$ 23.5 (max 85.8) $PM_{10-2.5}$ NR
Vedal et al., 1998 Port Alberni, CAN (children)	PEF -1.35 (-2.7, -0.05)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

* Studies in italics available in 1996 CD
** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 3. ESTIMATED CARDIOVASCULAR MORBIDITY EFFECTS PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Study Location*	% increase (95% CI) per 50 $\mu g/m^3$ PM_{10} Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	% increase (95% CI) per 25 $\mu g/m^3$ $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Increased Hospitalization				
Total Cardiovascular:				
Samet et al., 2000 14 U.S. Cities (>65 years)	6.0 (5.1, 6.8)	---	---	PM_{10} means 24.4-45.3
Schwartz, 1999 8 U.S. Counties (>65 years)	5.0 (3.7, 6.4)	---	---	PM_{10} means 23-37
Linn et al., 2000 Los Angeles, CA (>29 years)	3.25 (2.04, 4.47)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar, 2000b Cook Co., IL (>65 years)	4.2 (3.0, 5.5)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000b Los Angeles, CA (>65 years)	3.3 (2.0, 4.5)	(65+) 4.30 (2.52, 6.11) (<65) 3.54 (1.83, 5.27)	---	PM_{10} median 44, 7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2000b Maricopa Co., AZ (>65 years)	-2.4 (-6.9, 2.3)	---	---	PM_{10} median 41 (9, 252)
Morris and Naumova, 1998 Chicago, IL (>65 years)	3.92 (1.02, 6.90)	---	---	PM_{10} 41 (6, 117)
Schwartz, 1997 Tucson, AZ (>65 years)	6.07 (1.12, 1.27)	---	---	PM_{10} 42 (90% 63)
Gwynn et al., 2000 Buffalo, NY (all ages)	5.69 (-3.29, 15.50)	1.35 (-1.14, 4.28) (15 $\mu g/m^3$ $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) $nmol/m^3$
Tolbert et al., 2000a Atlanta, GA (all ages)	5.1 (-7.9, 19.9)	6.11 (-3.08, 16.17)	17.63 (-4.63, 45.07)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)

Stieb et al., 2000 St. John, CAN (all ages)	39.2 (5.0, 84.4)	15.11 (0.61, 11.03)	---	summer 93 PM_{10} 14.0 (max 70.3) $PM_{2.5}$ 8.5 (max 53.2)
Burnett et al., 1997 Toronto, CAN (all ages)	12.07 (1.43, 23.81)	7.18 (-0.61, 15.60)	20.46 (8.24, 34.06)	PM_{10} 28.4 (4, 102) $PM_{2.5}$ 16.8 (1, 66) $PM_{10-2.5}$ 11.6 (1, 56)
Ischemic Heart Disease:				
Schwartz and Morris 1995 *Detroit, MI*	*2.83 (0.72, 4.98)*	---	---	*PM_{10} 48 (22-82)***
Lippmann et al., 2000 Detroit, MI (>65 years)	8.91 (0.51, 18.03)	4.33 (-1.39, 10.39)	10.54 (2.73, 18.95)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	8.56 (5.33, 11.48)	8.05 (5.38, 10.78)	3.74 (1.30, 6.25)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Dysrhythmias:				
Tolbert et al., 2000a Atlanta, GA (all ages)	13.41 (-14.08, 48.99)	6.11 (-12.63, 28.86)	53.16 (2.07, 129.81)	$PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Lippmann et al., 2000 Detroit, MI (>65 years)	2.94 (-6.77, 13.65)	3.24 (-6.54, 14.04)	0.21 (-12.25, 14.43)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	8.41 (2.89, 14.23)	6.06 (1.94, 10.35)	5.13 (-0.21, 10.75)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Heart Failure:				
Schwartz and Morris, 1995 *Detroit, MI*	*5.04 (1.91, 8.27)*	---	---	*PM_{10} 48 (22-82)****
Linn et al., 2000 Los Angeles, CA (>29 years)	2.02 (-0.94, 5.06)	---	---	PM_{10} 45.5 (5, 132)

Lippmann et al., 2000 Detroit, MI (>65 years)	9.70 (0.17, 20.13)	9.06 (2.36, 16.19)	5.21 (-3.29, 14.46)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	9.70 (4.17, 15.52)	6.59 (2.50, 10.83)	7.88 (2.28, 13.78)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Myocardial Infarction:				
Linn et al., 2000 Los Angeles, CA (>29 years)	3.04 (0.06, 6.12)	---	---	PM_{10} 45.5 (5, 132)
Cardiac arrhythmia:				
Linn et al., 2000 Los Angeles, CA (>29 years)	1.01 (-1.93, 4.02)	---	---	PM_{10} 45.5 (5, 132)
Cerebrovascular:				
Linn et al., 2000 Los Angeles, CA (>29 years)	0.30 (-2.13, 2.79)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar, 2000b Cook Co., IL (>65 years)	3.22 (1.46, 5.03)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000b Los Angeles, CA(>65 years)	1.00 (-1.78, 3.86)	1.51 (-0.76, 3.82)	---	$PM_{2.5}$ 22 (4, 86) $PM_{10-2.5}$ ---
Moolgavkar, 2000b Maricopa Co., AZ (>65 years)	1.00 (-8.40, 11.38)	---	---	PM_{10} median 41 (9, 252)
Burnett et al., 1999 Toronto, CAN (all ages)	"NEG" reported	"NEG" reported	"NEG" reported	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Peripheral circulation diseases:				
Burnett et al., 1999 Toronto, CAN (all ages)	2.58 (-2.67, 8.11)	"NEG" reported	5.63 (0.32, 11.23)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Stroke:				

Linn et al., 2000 Los Angeles, CA (>29 years)	6.72 (3.64, 9.90)	---	---	PM_{10} 45.5 (5, 132)
Lippmann et al., 2000 Detroit, MI (>65 years)	4.80 (-5.47, 16.19)	1.80 (-5.30, 9.43)	4.90 (-4.69, 15.45)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)

* Studies in italics available in 1996 CD
** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 4. Data used in creating Figures 3-4 through 3-9. Effect estimates and confidence intervals for PM-mortality and morbidity associations, and data for number of study days, number of health events per day, and the product of the number of days and number of events.

(A) PM_{10}-mortality associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Samet et al., 2000, 90 U.S. city, total	2.30	0.10	4.50	**	**	1588776	14.278474
Samet 20-city total	2.58	0.41	4.79			1051794.5	13.866008311
Samet et al., 2000, 20 U.S. city, cardiorespiratory	3.45	1.01	5.94	**	**	577275.5	13.2660749012
Schwartz 2000, Chicago, total	4.53	3.11	5.96	2190	132	289080	12.574459
Styer et al., 1995, Chicago, total	4.08	0.08	8.24	2190	117	256230	12.453831
Burnett et al., 1998, Toronto, total	3.46	1.74	5.21	5475	40.17	219930.75	12.301068
Moolgavkar et al., 2000, LA, cardiovascular	4.47	1.65	7.37	3285	57	187245	12.140173
Ito and Thurston, 1996, Chicago, total	2.47	1.26	3.69	1529	116.5	178128.5	12.09026
Moolgavkar et al., 2000, Cook Co, cardiovascular	2.21	0.37	4.09	3285	43	141255	11.858322
Burnett et al., 2000, 8 Canadian cities, total	1.74	0.52	2.97	**	**	112102.6	11.62717
Ito and Thurston, 1996, Chicago, circulatory	1.49	-0.72	3.74	1529	56.2	85929.8	11.361286
Schwartz et al., 1996, St. Louis, total	3.04	0.76	5.37	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston, total	6.15	3.56	8.80	1140	60.2	68628	11.136456
Kinney et al., 1995, LA, total	2.47	-0.17	5.18	364	153	55692	10.927592
Moolgavkar et al., 2000, Maricopa, cardiovascular	8.85	2.67	15.39	3285	13	42705	10.662071
Pope et al., 1999, Salt Lake City, total	2.33	0.05	4.66	3700	11.32	41884	10.642659
Schwartz et al., 1996, Knoxville, total	4.58	0.27	9.08	1481	14.2	21030.2	9.9537148
Moolgavkar et al., 2000, LA, COPD	5.90	-1.64	14.03	3285	6	19710	9.8888814
Schwartz et al., 1996, Portage, total	3.55	-1.71	9.09	1436	13.6	19529.6	9.8796865
Schwartz et al., 1993, Birmingham, total	5.36	1.16	9.73	1087	17.1	18587.7	9.8302554
Lippmann et al., 2000, Detroit, total	4.41	-0.98	10.10	344	53	18232	9.8109336
Pope et al., 1999, Salt Lake City, cardiovascular	6.50	2.21	10.98	3700	4.72	17464	9.7678969
Ostro et al., 2000, Coachella Valley, total	2.01	-0.99	5.10	3011	5.8	17463.8	9.7678854
Fairley, 1999, Santa Clara, total	8.00	3.55	12.65	823	20	16460	9.7086885
Ito and Thurston, 1996, Chicago, respiratory	6.77	1.97	11.79	1529	9.8	14984.2	9.6147516
Moolgavkar et al., 2000, Cook Co., COPD	5.39	0.30	10.74	3285	4	13140	9.4834163
Pope et al., 1999, Provo/Orem, total	1.87	-2.14	6.04	3687	2.65	9770.55	9.187128
Gwynn et al., 2000, Buffalo, total	12.33	2.50	23.11	175	54	9450	9.15377
Mar et al., 2000, Phoenix, total	5.44	0.06	11.12	1095	8.55	9362.25	9.1444409
Lippmann et al., 2000, Detroit, circulatory	6.86	-1.28	15.66	344	25	8600	9.0595175

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Ostro et al., 2000, Coachella Valley, cardiovascular	6.09	2.05	10.29	3011	2.7	8129.7	9.0032793
Moolgavkar et al., 2000, Maricopa, COPD	8.08	-4.58	22.41	3285	2	6570	8.7902691
Schwartz et al., 1996, Topeka, total	-2.48	-9.33	4.90	1432	4.5	6444	8.7709047
Ostro et al., 1999, Coachella Valley, total	4.60	0.58	8.78	1188	5.4	6415.2	8.7664255
Pope et al., 1999, Ogden, total	12.02	4.49	20.09	2308	2.55	5885.4	8.68023
Tsai et al., 2000, Newark NJ, total (PM15)	5.65	4.62	6.69	156	37	5772	8.6607739
Schwartz et al., 1996, Steubenville, total	4.58	0.76	8.54	1520	3.6	5472	8.6073995
Pope et al., 1992, Utah Valley, total	7.63	4.41	10.95	1706	2.7	4606.2	8.4351585
Pope et al., 1999, Provo/Orem, cardiovascular	8.60	2.40	15.18	3687	1.17	4313.79	8.3695721
Mar et al., 2000, Phoenix, cardiovascular	9.86	1.91	18.42	1095	3.85	4215.75	8.3465828
Pope et al., 2000, Salt Lake City, respiratory	8.17	-0.97	18.14	3700	0.96	3552	8.1752661
Gwynn et al., 2000, Buffalo, circulatory	17.83	0.69	37.88	175	19	3325	8.109225
Tsai et al., 2000, Newark NJ, cardiorespiratory	7.79	3.64	12.10	156	21	3276	8.0943784
Pope et al., 1999, Ogden, cardiovascular	1.41	-8.33	12.18	2308	1.14	2631.12	7.8751649
Ostro et al., 1999 , Coachella Valley, cardiovascular	8.33	2.14	14.89	1188	1.8	2138.4	7.6678132
Pope et al., 1992, Utah Valley, cardiovascular	9.36	1.91	17.36	1706	1.24	2115.44	7.6570181
Tsai et al., 2000, Elizabeth NJ, total	-4.88	-17.88	10.19	156	13	2028	7.6148054
Tsai et al., 2000, Camden NJ, total	11.07	0.70	22.51	156	11	1716	7.4477513
Ostro et al., 2000, Coachella Valley, respiratory	-1.99	-11.41	8.44	3011	0.52	1565.72	7.3561011
Lippmann et al., 2000, Detroit, respiratory	7.84	-10.18	29.47	344	4	1376	7.226936
Tsai et al., 2000, Elizabeth NJ, cardiorespiratory	3.05	-11.04	19.36	156	7	1092	6.9957662
Pope et al., 1999, Provo/Orem, respiratory	2.22	-9.83	15.89	3687	0.27	995.49	6.9032351
Tsai et al., 2000, Camden NJ, cardiorespiratory	15.03	4.29	26.87	156	6	936	6.8416155
Gwynn et al., 2000, Buffalo, respiratory	17.89	-14.87	63.25	175	5	875	6.7742239
Ostro et al., 1999, Coachella Valley, respiratory	13.88	3.25	25.61	1188	0.6	712.8	6.5692009
Pope et al., 1999, Ogden, respiratory	23.80	2.77	49.14	2308	0.26	600.08	6.397063
Pope et al., 1992, Utah Valley, respiratory	19.78	3.51	38.61	1706	0.27	460.62	6.1325734

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(B) $PM_{2.5}$-Mortality Associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Burnett et al., 1998, Toronto, total	4.79	3.26	6.34	5475	40.17	219930.75	12.301068
Moolgavkar et al., 2000, LA, cardiovascular	2.59	0.38	4.85	3285	57	187245	12.140173
Schwartz 2000, Boston, total	5.33	1.81	8.98	2920	60	175200	12.073683

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Goldberg et al., 2000, Montreal, total	5.81	3.36	8.32	3653	38.6	141005.8	11.856556
Burnett et al., 2000, 8 Canadian cities, total	3.03	1.10	4.99	**	**	117452	11.673785
Ostro et al., 1995, So. California, total	0.28	-0.61	1.17	2555	40.73	104065.15	11.552772
Schwartz et al., 1996, St. Louis, total	2.77	1.13	4.44	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston, total	5.59	3.80	7.41	1140	60.2	68628	11.136456
Goldberg et al., 2000, Montreal, cardiovascular	3.48	-0.16	7.26	3653	15.7	57352.1	10.956965
Ostro et al., 1995, So. California, circulatory	0.69	-0.35	1.74	2555	18.74	47880.7	10.776468
Schwartz et al., 1996, Knoxville, total	3.54	0.52	6.65	1481	14.2	21030.2	9.9537148
Moolgavkar et al., 2000, LA, COPD	2.67	-3.38	9.10	3285	6	19710	9.8888814
Schwartz t al., 1996, Portage, total	3.03	-0.84	7.05	1436	13.6	19529.6	9.8796865
Lippmann et al., 2000, Detroit, total	3.10	-0.63	6.98	344	53	18232	9.8109336
Goldberg et al., 2000, Montreal, respiratory	21.65	12.95	31.01	3653	3.1	11324.3	9.3347061
Ostro et al., 1995, So. California, respiratory	2.08	-0.35	4.51	2555	3.83	9785.65	9.1886723
Mar et al., 2000, Phoenix, total	3.03	-0.69	6.88	1095	8.55	9362.25	9.1444409
Lippmann et al., 2000, Detroit, circulatory	3.17	-2.29	8.94	344	25	8600	9.0595175
Fairley, 1999, Santa Clara, total	8.48	3.38	13.84	408	20	8160	9.0069994
Schwartz et al., 1996, Topeka, total	2.01	-4.83	9.35	1432	4.5	6444	8.7709047
Ostro et al., 2000, Coachella Valley, total	11.51	0.21	24.09	1041	5.8	6037.8	8.705795
Tsai et al., 2000, Newark NJ, total	4.34	2.82	5.89	156	37	5772	8.6607739
Schwartz et al., 1996, Steubenville, total	2.52	-0.24	5.35	1520	3.6	5472	8.6073995
Mar et al., 2000, Phoenix, cardiovascular	18.68	5.72	33.23	1095	3.85	4215.75	8.3465828
Tsai et al., 2000, Newark NJ, cardiorespiratory	5.13	3.09	7.21	156	21	3276	8.0943784
Ostro et al., 2000, Coachella Valley, cardiovascular	8.56	-6.35	25.84	1041	2.7	2810.7	7.9411888
Tsai et al., 2000, Elizabeth NJ, total	1.77	-5.45	9.53	156	13	2028	7.6148054
Tsai et al., 2000, Camden NJ, total	5.65	0.11	11.51	156	11	1716	7.4477513
Lippmann et al., 2000, Detroit, respiratory	2.28	-10.31	16.63	344	4	1376	7.226936
Tsai et al., 2000, Elizabeth NJ, cardiorespiratory	2.28	-4.97	10.08	156	7	1092	6.9957662
Tsai et al., 2000, Camden NJ, cardiorespiratory	6.18	0.61	12.06	156	6	936	6.8416155
Ostro et al., 2000, Coachella Valley, respiratory	-13.28	-43.05	32.06	1041	0.52	541.32	6.2940106

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(C) $PM_{10-2.5}$-Mortality Associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Burnett et al., 2000, 8 Canadian cities, total	1.82	-0.72	4.43	**	**	112186.7	11.62792

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Schwartz et al., 1996, St. Louis, total	0.50	-1.73	2.78	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston., total	0.50	-1.73	2.78	1140	60.2	68628	11.136456
Schwartz et al., 1996, Knoxville, total	2.52	-1.46	6.66	1481	14.2	21030.2	9.9537148
Schwartz et al., 1996, Portage, total	1.25	-3.06	5.76	1436	13.6	19529.6	9.8796865
Lippmann et al., 2000, Detroit, total	3.96	-1.22	9.42	344	53	18232	9.8109336
Ostro et al., 2000, Coachella Valley, total	1.28	-0.63	3.22	2990	5.8	17342	9.7608866
Lippmann et al., 2000, Detroit, respiratory	7.41	-9.07	26.87	344	25	8600	9.0595175
Fairley, 1999, Santa Clara, total	4.53	-6.66	17.05	408	20	8160	9.00699944796
Ostro et al., 2000, Coachella Valley, circulatory	2.56	0.66	4.49	2990	2.7	8073	8.9962804
Mar et al., 2000, Phoenix, total	2.97	-0.50	6.56	300	22.9	6870	8.8349194
Schwartz et al., 1996, Topeka, total	-3.22	-7.89	1.69	1432	4.5	6444	8.7709047
Schwartz et al., 1996, Steubenville, total	6.11	1.30	11.15	1520	3.6	5472	8.6073995
Mar et al., 2000, Phoenix, cardiovascular	6.45	1.42	11.73	1095	3.85	4215.75	8.3465828
Ostro et al., 2000, Coachella Valley, respiratory	-1.27	-6.24	3.95	2990	0.52	1554.8	7.3491022
Lippmann et al., 2000, Detroit, circulatory	7.82	0.03	16.23	344	4	1376	7.226936

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(D) Associations between PM_{10} and admissions to the hospital or emergency room

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Linn et al., 2000, LA, respiratory	2.89	1.09	4.72	3640	207	753480	13.532458
Burnett et al., 1999, Toronto, respiratory	14.20	9.32	19.30	5475	13	71175	11.172897
Gwynn et al., 2000, Buffalo, respiratory	3.14	-1.78	8.31	812	56.3	45715.6	10.730195
Schwartz et al., 1996, Cleveland, respiratory	5.83	0.54	11.40	1095	22	24090	10.089552
Moolgavkar et al., 1997, Minn/St. Paul, respiratory	8.72	4.59	13.01	1979	10.55	20878.45	9.9464728
Moolgavkar, et al., 1997, Birmingham, respiratory	1.51	-1.43	4.54	2098	8.26	17329.48	9.7601644
Stieb et al., 2000, St. John, respiratory	8.84	1.84	16.32	1260	10.9	13734	9.5276298
Burnett et al., 1997,Toronto, respiratory	6.95	2.91	11.15	388	23.7	9195.6	9.1264804
Schwartz et al., 1995, New Haven, respiratory	6.00	-0.28	12.68	1095	8.1	8869.5	9.0903737
Schwartz et al., 1995, Tacoma, respiratory	10.00	3.21	17.24	1095	4.2	4599	8.4335942
Schwartz et al., 1996, Spokane, respiratory	8.50	3.61	13.62	821	3.9	3201.9	8.0714997
Delfino et al., 1993, Montreal, respiratory	40.49	11.25	77.43	92	20.12	1851.04	7.5235029
Thurston et al., 1994 Toronto, respiratory	23.26	2.03	44.49	**	**	1693	7.43425738213
Moolgavkar, 2000c, LA, COPD	6.09	1.09	11.34	3285	20	65700	11.092854
Samet et al., 2000b, 14 U.S. Cities, COPD	10.30	7.68	12.98	**	**	60683.31	11.013424

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Moolgavkar, 2000c, Cook Co,, COPD	2.41	-0.21	5.11	3285	12	39420	10.582029
Burnett et al., 1999, Toronto, COPD	6.90	1.32	12.78	5475	5	27375	10.217385
Moolgavkar, 2000c, Maricopa Co., COPD	6.92	-4.15	19.25	3285	4	13140	9.4834163
Schwartz, 1994, Detroit, COPD	10.63	4.41	17.21	1191	5.8	6907.8	8.8404065
Moolgavkar et al., 1997, Minn/St. Paul, COPD	6.89	-0.64	14.99	1979	2.91	5758.89	8.6585
Moolgavkar et al., 2000, King Co., COPD	15.93	5.2	27.75	2022	2.33	4711.26	8.4577107
Lippmann et al., 2000, Detroit, COPD	9.60	-5.28	26.82	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	-3.45	-33.01	29.92	350	9.7	3395	8.130059
Chen et al., 2000, Reno, COPD	9.41	2.20	17.12	1815	1.72	3121.8	8.046165
Schwartz, 1994, Birmingham, COPD	12.69	3.81	22.34	1369	2.2	3011.8	8.0102932
Schwartz, 1994, Minn/St. Paul, COPD	25.30	9.47	43.42	1251	2.2	2752.2	7.9201559
Schwartz, et al., 1996, Spokane, COPD	17.10	7.85	27.14	821	1	821	6.7105231
Burnett et al., 1999, Toronto, asthma	8.88	3.65	14.36	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	13.70	5.46	22.58	2920	2.7	7884	8.9725907
Tolbert et al., 2000a, Atlanta, asthma	13.24	1.21	26.70	276	22	6072	8.7114433
Tolbert et al., 2000b, Atlanta, asthma	18.77	-8.65	54.42	350	15.8	5530	8.6179431
Lipsett et al., 1997, Santa Clara, asthma	9.09	2.72	15.85	368	7.6	2796.8	7.9362312
Nauenberg and Basu, 1999, LA, asthma	20.02	5.33	34.71	315	8.74	2753.1	7.9204828
Choudbury et al., 1997, Anchorage, asthma	20.72	11.65	29.79	1095	2.42	2649.9	7.8822772
Norris et al., 2000, Spokane, asthma	2.35	-10.93	17.61	816.7	3.2	2613.44	7.8684226
Norris et al., 1999, Seattle, asthma	75.91	25.08	147.39	468.5	1.9	890.15	6.79139
Norris et al., 1998, Seattle, asthma	56.20	10.38	121.06	487	1.8	876.6	6.7760508
Samet et al., 2000b, 14 U.S. cities, pneumonia	10.30	7.70	13.00	**	**	168894.37	12.037029
Schwartz, 1994, Detroit, pneumonia	5.92	1.95	10.05	1191	15.7	18698.7	9.8362093
Moolgavkar et al., 1997, Minn/St. Paul, pneumonia	3.54	-0.49	7.72	1979	7.64	15119.56	9.6237445
Schwartz, 1994, Birmingham, pneumonia	9.09	3.51	14.97	1369	5.9	8077.1	8.9967882
Schwartz, 1994, Minn/St. Paul, pneumonia	8.17	1.22	15.59	1251	6	7506	8.923458
Lippmann et al., 2000, Detroit, pneumonia	21.43	8.18	36.29	490	12	5880	8.679312
Schwartz et al., 1996, Spokane, pneumonia	5.30	-1.51	12.58	821	1.9	1559.9	7.352377
Linn et al., 2000, LA, cardiovascular	3.25	2.04	4.47	3640	428	1557920	14.258862
Samet et al., 2000b, 14 U.S. cities, cardiovascular	5.99	5.15	6.83	**	**	673571.53	13.420349
Moolgavkar, 2000b, LA, cardiovascular	3.23	1.17	5.32	3285	172	565020	13.244616
Moolgavkar, 2000b, Cook Co., cardiovascular	4.24	3.00	5.50	3285	110	361350	12.797602
Moolgavkar, 2000b, Maricopa Co., cardiovascular	-2.39	-6.90	2.35	3285	33	108405	11.593629
Gwynn et al., 2000, Buffalo, cardiovascular	10.98	3.79	18.66	812	83	67396	11.118341
Schwartz et al., 1999, 8 US Counties, cardiovascular	5.02	3.67	6.39	1095	31.5	34492.5	10.448497

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1997,Toronto, cardiovascular	7.66	0.93	14.84	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	5.10	-7.88	19.91	350	45.1	15785	9.6668154
Schwartz, 1997, Tucson, cardiovascular	6.07	1.12	11.27	829.9	13.4	11120.66	9.3165599
Stieb et al., 2000, St. John, cardiovascular	32.51	10.20	59.34	1260	3.5	4410	8.39163
Burnett et al., 1999, Toronto, ischemic heart disease	8.36	5.33	11.48	5475	24	131400	11.786001
Schwartz and Morris, 1995, Detroit, ischemic heart disease	2.83	0.72	4.98	1191	44.1	52523.1	10.869008
Lippmann et al., 2000, Detroit, ischemic heart disease	8.91	0.51	18.03	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	8.41	2.89	14.23	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	13.14	-14.08	48.99	350	11.2	3920	8.2738469
Lippmann et al., 2000, Detroit, dysrhythmia	2.94	-6.76	13.65	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	9.70	4.17	15.52	5475	9	49275	10.805172
Morris et al., 1995, Chicago, CHD/heart failure	3.92	1.02	6.90	1168	34	39712	10.589409
Schwartz and Morris, 1995, Detroit, CHD/heart failure	5.04	1.91	8.27	1191	26.2	31204.2	10.348308
Lippmann et al., 2000, Detroit, CHD/heart failure	9.70	0.17	20.13	490	17	8330	9.0276187

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Samet et al., 2000b, Tables 7 and 9

(E) Associations between $PM_{2.5}$ and admissions to the hospital or emergency room

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1999, Toronto, respiratory	10.77	7.18	14.47	5475	13	71175	11.172897
Lumley and Heagerty, 1999, King Co., respiratory	5.92	1.10	10.97	2920	7.5	21900	9.9942419
Stieb et al., 2000, St. John, respiratory	5.69	0.62	11.02	1260	10.9	13734	9.5276298
Burnett et al., 1997, Toronto, respiratory	6.24	2.48	10.14	388	23.7	9195.6	9.1264804
Lippmann et al., 2000, Detroit , respiratory	12.51	3.69	22.08	490	12	5880	8.679312
Delfino et al., 1997, Montreal, respiratory	23.88	4.94	42.83	95	26.9	2555.5	7.8460032
Delfino et al., 1998, Montreal, respiratory	13.17	-0.22	26.57	92	20.12	1851.04	7.5235029
Thurston et al., 1994, Toronto, respiratory	15	2	28	**	**	1693	7.4342574
Moolgavkar, 2000c, LA, COPD	5.08	0.91	9.41	3285	20	65700	11.092854
Burnett et al., 1999, Toronto, COPD	4.78	-0.17	9.98	5475	5	27375	10.217385
Moolgavkar, et al., 2000, King Co.	6.40	0.90	12.10	3287	7.75	25474.25	10.145442
Lippmann et al., 2000, Detroit, COPD	5.49	-4.72	16.80	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	12.44	-7.88	37.24	350	9.7	3395	8.130059
Burnett et al., 1999, Toronto, asthma	6.45	2.47	10.57	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	8.66	3.29	14.32	2920	2.7	7884	8.9725907
Tolbert et al., 2000, Atlanta, asthma	2.27	-14.79	22.73	350	15.8	5530	8.6179431

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Norris et al., 1999, Seattle, asthma	44.50	21.70	71.40	487	1.8	876.6	6.7760508
Moolgavkar, 2000b, LA, cardiovascular	4.30	2.52	6.11	3285	172	565020	13.244616
Burnett et al., 1997, Toronto, cardiovascular	5.90	1.79	10.18	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	6.11	-3.07	16.16	350	45.1	15785	9.6668154
Stieb et al., 2000, St. John, cardiovascular	15.11	-0.25	32.82	1260	3.5	4410	8.39163
Burnett et al., 1999, Toronto, ischemic heart disease	8.05	5.38	10.78	5475	24	131400	11.786001
Lippmann et al., 2000, Detroit, ischemic heart disease	4.33	-1.39	10.39	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	6.06	1.94	10.35	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	6.11	-12.62	28.85	350	11.2	3920	8.2738469
Lippmann et al., 2000, Detroit, dysrhythmia	3.24	-6.54	14.04	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	6.59	2.50	10.83	5475	9	49275	10.805172
Lippmann et al., 2000, Detroit, CHD/heart failure	9.06	2.36	16.19	490	17	8330	9.0276187

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Table 1

(F) Associations between $PM_{10-2.5}$ and admissions to the hospital or emergency room

citation location, admissions category	study number	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1999, Toronto, respiratory	1	9.31	4.64	14.18	5475	13	71175	11.172897
Burnett et al., 1997, Toronto, respiratory	2	8.46	3.51	13.64	388	23.7	9195.6	9.1264804
Lippmann et al., 2000, Detroit, respiratory	3	11.90	0.65	24.41	490	12	5880	8.679312
Thurston et al., 1994, Toronto, respiratory	4	22.25	-9.53	54.03	**	**	1693	7.4342574
Moolgavkar, 2000b, LA, COPD	5	5.08	-0.44	10.90	3285	20	65700	11.092854
Burnett et al., 1999, Toronto, COPD	6	12.83	4.93	21.33	5475	5	27375	10.217385
Lippmann et al., 2000, Detroit, COPD	7	9.29	-4.19	24.66	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	8	-23.03	-50.68	20.12	350	9.7	3395	8.130059
Burnett et al., 1999, Toronto, asthma	9	11.05	5.75	16.62	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	10	11.12	2.83	20.08	2920	2.7	7884	8.9725907
Tolbert et al., 2000, Atlanta, asthma	11	21.08	-18.21	79.25	350	15.8	5530	8.6179431
Burnett et al., 1997, Toronto, cardiovascular	12	13.46	5.52	22.01	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	13	17.63	-4.61	45.05	350	45.1	15785	9.6668154
Burnett et al., 1999, Toronto, ischemic heart disease	14	3.74	1.30	6.25	5475	24	131400	11.786001
Lippmann et al., 2000, Detroit, ischemic heart disease	15	10.54	2.73	18.95	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	16	5.13	-0.21	10.75	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	17	53.16	2.15	129.65	350	11.2	3920	8.2738469

citation location, admissions category	study number	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Lippmann et al., 2000, Detroit, dysrhythmia	18	0.21	-12.25	14.43	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	19	7.88	2.28	13.78	5475	9	49275	10.805172
Lippmann et al., 2000, Detroit, CHD/heart failure	20	5.21	-3.29	14.46	490	71	34790	10.457085

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Table 1

APPENDIX B

FIGURES AND TABLES FOR CHAPTER 5, SECTION 5.2, ON VISIBILITY

FIGURES:

Figure 5-1 and 5-2 – In Staff Paper Text

Washington, DC Images

[See Figures 3 through 10 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file WASHDC8IMAGES. These images were generated using WinHaze 2.8.0.]

Chicago, IL Images

[See Figures 11 through 16 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file CHICAGO6IMAGES. These are actual photographs provided by Illinois EPA.]

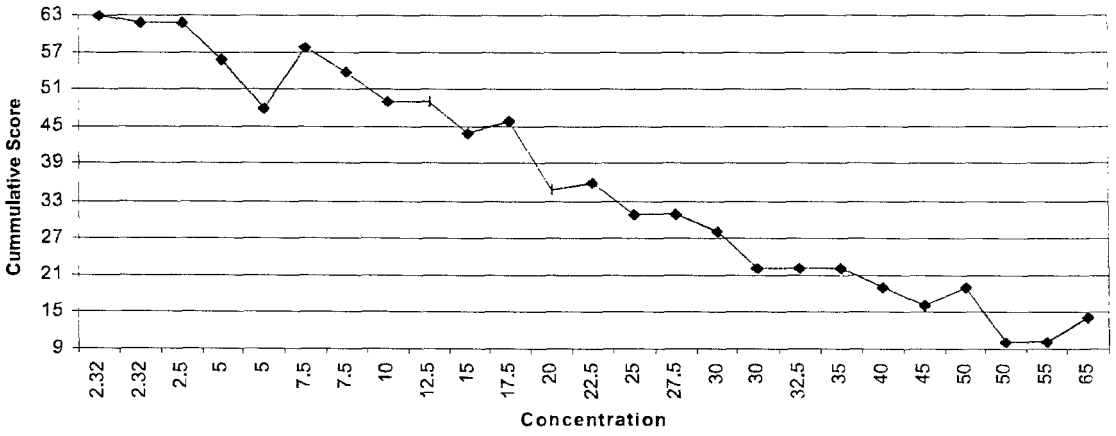

Figure 17. Rating of Visual Air Quality for Washington, DC Images. November 2000 Pilot Project.

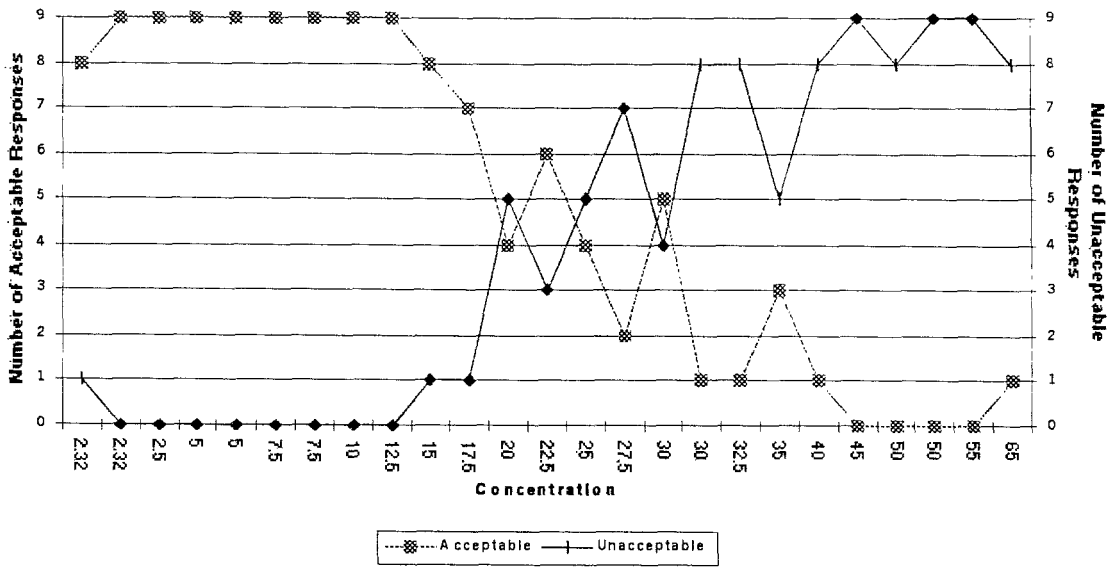

Figure 18. Rating of Acceptability / Unacceptability for Washington, DC Images. November 2000 Pilot Project.

Denver, Colorado Images

[See Figures 19 through 26 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file DENVER8IMAGES. These images were generated using WinHaze 2.8.0.]

Figure 19. Denver, CO – 35 Mm^{-1}
Figure 20. Denver, CO – 43 Mm^{-1}
Figure 21. Denver, CO – 51 Mm^{-1}
Figure 22. Denver, CO – 61 Mm^{-1}
Figure 23. Denver, CO – 76 Mm^{-1}
Figure 24. Denver, CO – 93 Mm^{-1}
Figure 25. Denver, CO – 167 Mm^{-1}
Figure 26. Denver, CO – 258 Mm^{-1}

Phoenix, Arizona Images

[See Figures 27 through 34 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file PHOENIX8IMAGES. These images were generated using WinHaze 2.8.0.]

Figure 27. Phoenix, AZ – 2.5 $\mu g/m^3$ $PM_{2.5}$
Figure 28. Phoenix, AZ – 5 $\mu g/m^3$ $PM_{2.5}$
Figure 29. Phoenix, AZ – 10 $\mu g/m^3$ $PM_{2.5}$
Figure 30. Phoenix, AZ – 15 $\mu g/m^3$ $PM_{2.5}$
Figure 31. Phoenix, AZ – 20 $\mu g/m^3$ $PM_{2.5}$
Figure 32. Phoenix, AZ – 30 $\mu g/m^3$ $PM_{2.5}$
Figure 33. Phoenix, AZ – 40 $\mu g/m^3$ $PM_{2.5}$
Figure 34. Phoenix, AZ – 65 $\mu g/m^3$ $PM_{2.5}$

TABLES:

Table 1. Aerosol Concentrations Used to Create Washington, DC Images.

Percent of Fine Mass		Sulfate: 50%	Nitrate: 10%	OC: 25%	EC: 10%	Soil: 5%	Coarse: 30% x fine mass
Slide	Image (ug/m3)	Sulfate (ug/m3)	Nitrate (ug/m3)	OC (ug/m3)	EC (ug/m3)	Soil (ug/m3)	Coarse (ug/m3)
1	65.0	32.50	6.50	16.25	6.50	3.25	19.50
2	60.0	30.00	6.00	15.00	6.00	3.00	18.00
3	55.0	27.50	5.50	13.75	5.50	2.75	16.50
4	52.5	26.25	5.25	13.13	5.25	2.63	15.75
5	50.0	25.00	5.00	12.50	5.00	2.50	15.00
6	47.5	23.75	4.75	11.88	4.75	2.38	14.25
7	45.0	22.50	4.50	11.25	4.50	2.25	13.50
8	42.5	21.25	4.25	10.63	4.25	2.13	12.75
9	40.0	20.00	4.00	10.00	4.00	2.00	12.00
10	37.5	18.75	3.75	9.38	3.75	1.88	11.25
11	35.0	17.50	3.50	8.75	3.50	1.75	10.50
12	32.5	16.25	3.25	8.13	3.25	1.63	9.75
13	30.0	15.00	3.00	7.50	3.00	1.50	9.00
14	27.5	13.75	2.75	6.88	2.75	1.38	8.25
15	25.0	12.50	2.50	6.25	2.50	1.25	7.50
16	22.5	11.25	2.25	5.63	2.25	1.13	6.75
17	20.0	10.00	2.00	5.00	2.00	1.00	6.00
18	17.5	8.75	1.75	4.38	1.75	0.88	5.25
19	15.0	7.50	1.50	3.75	1.50	0.75	4.50
20	12.5	6.25	1.25	3.13	1.25	0.63	3.75
21	10.0	5.00	1.00	2.50	1.00	0.50	3.00
22	7.50	3.75	0.75	1.88	0.75	0.38	2.25
23	6.25	3.13	0.63	1.56	0.63	0.31	1.88
24	5.00	2.50	0.50	1.25	0.50	0.25	1.50
25	3.75	1.88	0.38	0.94	0.38	0.19	1.13
26	2.50	1.25	0.25	0.63	0.25	0.13	0.75
27	2.32 (natural) *	0.20	0.10	1.50	0.02	0.50	3.00

* Note: For slide 27, NO2 = 0.0 ppb

Table 2. Visibility Parameters for Washington, DC Images.

Slide	$PM_{2.5}$ (ug/m3)	Light Extinction (Mm-1)	Visual Range (km)	Deciviews
1	65.0	507	7.7	39.3
2	60.0	469	8.3	38.5
3	55.0	431	9.1	37.6
4	52.5	412	9.5	37.2
5	50.0	393	10.0	36.7
6	47.5	374	10.5	36.2
7	45.0	355	11.0	35.7
8	42.5	336	11.6	35.1
9	40.0	317	12.3	34.6
10	37.5	298	13.1	33.9
11	35.0	279	14.0	33.3
12	32.5	260	15.0	32.6
13	30.0	241	16.2	31.8
14	27.5	222	17.6	31.0
15	25.0	203	19	30.1
16	22.5	184	21	29.1
17	20.0	165	24	28.0
18	17.5	146	27	26.8
19	15.0	127	31	25.4
20	12.5	108	36	23.8
21	10.0	89	44	21.9
22	7.50	70	56	19.5
23	6.25	61	64	18.0
24	5.00	51	76	16.3
25	3.75	42	94	14.3
26	2.50	32	122	11.7
27	2.32 (natural)	21	185	7.5